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BUREAU OF HYGIENE AND TROPICAL DISEASES.

TROPICAL DISEASES BULLETIN.

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THE TREATMENT OF MALARIA.

A Review of Some Recent Papers.

By Norman White, C.I.E., M.D., D.P.H., I.M.S. (retd.).

The treatment of malaria is a subject of perennial interest. This in itself is an indication that finality has not yet been reached. Numerous synthetic remedies have proved their value but none satisfies all requirements. Cinchona alkaloids are still almost indispensable. The relative value of antimalarial drugs in different circumstances and the manner in which they can best be employed are matters of outstanding urgency and importance at the present time. British Empire and Allied forces are operating in many endemic and hyperendemic malarial regions, many allied ships are calling at malaria-infested ports, and Java, which produced nine-tenths of the quinine in the world's markets, is in enemy hands. It is interesting to reflect how serious the situation might have been had Japan's aggressive designs been accomplished before Germany's pioneer research work had given us atebrin.

The majority of papers dealing with the treatment of malaria have certain features in common ; their authors all know how malaria can best be treated. There is a praiseworthy dogmatism about their pronouncements but there is anything but uniformity of dogma. There is not even uniformity of opinion regarding the manner in which quinine, the oldest and best tried remedy of all, should be administered. There is still less uniformity of opinion regarding the merits and demerits of the different remedies, the indications for their use, and the doses which can be safely administered in the treatment of the acute attack of malaria and in clinical prophylaxis. In great part the diversity of views is explained by the very different malarial conditions prevailing in the countries in which practical experience has been gained, by differences in strains of malaria parasites, by differences in the manner in which different races of mankind react to infection and to some of the specific remedies, and by differing degrees of malarial endemicity.

During the decade before this war the Malaria Commission of the League of Nations arranged for large scale observations in many different parts of the world to determine the relative value of the

cinchona alkaloids and the synthetic remedies in the treatment and in the clinical prophylaxis of malaria. Short courses of treatment were in general advocated. None of the specifics are capable of procuring *therapia sterilisans magna*. In endemic regions where reinfections could hardly be avoided a certain residual latent infection appeared to be of value in building up a state of resistance or premunition. Recrudescences and relapses could be treated with repeated short courses as they appeared. In hyperendemic areas in which the adult native population had acquired a solid immunity against local strains of malaria, there was an increasing consensus of opinion that specific remedies should be used but sparingly, little more than enough to tide infants and young children over the dangers inherent in the process of acquiring such immunity by repeated infections. For susceptible sojourners in such hyperendemic lands reliance was placed in the regular administration of specific remedies in prophylactic doses, most commonly quinine, 5 grains a day.

Such considerations are applicable for the most part only to civilian populations engaged in peace-time pursuits. In war other considerations predominate. Then it is necessary to keep as many men as possible fighting fit, for the most part highly susceptible individuals exposed to the risk of repeated infections and the stress and strain of war in a trying climate.

It was appropriate that the Royal Society of Tropical Medicine and Hygiene should have devoted a meeting to the consideration of the treatment of malaria. In an interesting paper CHRISTOPHERS[1] surveyed the whole field of the specific medication of malaria with the cinchona derivatives and the synthetic drugs. He stigmatized as malpraxis the intramuscular injection of quinine; the intramuscular injection of atebrin was not so harmful. In atebrin (mepacrine) "we have a substitute, and, at the present time, the only substitute, for quinine."

HUGHES[2] confined himself to a consideration of the treatment of malaria in a hyperendemic zone. In hyperendemic zones, as contrasted with endemic zones, *P. falciparum* malaria is predominant; severe and fatal cases among the indigenous population are confined to childhood; the native population, having acquired an immunity, flourishes while newcomers are rapidly killed off unless protected or treated; an epidemic cannot occur among the native population; antilarval measures may do more harm than good by diminishing the repeated infections on which the immunity depends. The author considers that the vast majority of clinical attacks of malaria in adult natives of hyperendemic areas require no quinine or other specific treatment; the disease runs a mild course and terminates naturally. The presence of malarial parasites in the blood of adult West Africans is no indication for giving quinine; 48·6 per cent. of the adult natives of Southern Nigeria show malarial parasites in their blood. Native children, however, may need specific treatment; many cases require injections of quinine. Europeans in these hyperendemic areas are exposed

[1] CHRISTOPHERS (S. Rickard). The Treatment of Malaria and Some Points about the Drugs in Use against this Disease.—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1942. Aug. 31. Vol. 36. No. 2. pp. 49–59.

[2] HUGHES (William). The Treatment of Malaria in a Hyperendemic Zone. *Trans. Roy. Soc. Trop. Med. & Hyg.* 1942. Aug. 31. Vol. 36. No. 2. pp. 60–66. Discussion pp. 66–74 [YORKE (Warrington), HILL (T. Rowland), MANSON-BAHR (Philip) & CHRISTOPHERS (S. Rickard)].

to great dangers; prophylactic ingestion of 5 grains of quinine a day is almost universal among them. This medication has its drawbacks but it has stood the test of time. In the treatment of attacks of malaria 30 grains of quinine a day are given during the acute stage, and a maintenance dose of about five grains a day. Injections of quinine are frequently required and intramuscular injections have advantages. There are no empirical findings to show that intravenous injections are superior to intramuscular injections. In comatose cases the intramuscular injection of quinine together with the intravenous injection of saline and glucose is remarkably successful. In all cases fever should be treated promptly; if quinine is not given promptly incessant vomiting and coma may develop. When the European leaves the hyperendemic zone treatment is advisable. For this the author has recently recommended a course of atebrin and quinine to be begun 10 days after leaving—2 tablets [each of 0·1 gm.] of atebrin and 2 five-grain tablets of quinine daily for 7 days. In the treatment of blackwater fever, alkali therapy is a *sine qua non*; the urine should be rendered alkaline within 24 hours.

Warrington YORKE expects a greatly increased demand for antimalarial drugs and stresses the importance of the manufacture of an adequate quantity of mepacrine (atebrin) and the conservation of existing stocks of quinine by its economic and efficient use in the treatment of malaria. In the treatment of acute simple tertian malaria nothing is gained by giving more than 20–30 grains of quinine a day in the acute stage: as a suppressive treatment, interrupted is preferable to continuous quinine administration. The quinine treatment of malaria in Liverpool consists of the administration of 30 grains of quinine in solution by mouth, daily for 4 consecutive days and thereafter 20 grains every Saturday and Sunday for 8 weeks, or longer should a relapse occur. It is imperatively necessary to control acute attacks of malaria among an unacclimatized population and should there be a shortage of antimalarial drugs it is the suppressive treatment that would have to be reduced. A treatment suggested by Army medical authorities consists of 30 grains of quinine daily for 6 days, 20 grains daily for four days, interval 7 days, and 20 grains daily for 7 days—400 grains of quinine in all. Even these quantities of quinine will not abolish the infection; three-quarters of this quinine is wasted. Intramuscular injections of quinine are sometimes invaluable.

Yorke also referred to the Standard Army Treatment of Malaria which consists of quinine 30 grains daily for 2 days; mepacrine 0·3 gm. daily for 5 days; two days' interval; pamaquin (plasmoquine) 0·03 gm. daily for 5 days. He asked what the pamaquin thus administered was supposed to do; was there any evidence that it reduced the relapse rate? The question is important as the drug is difficult to manufacture.

Rowland HILL's[3] contribution to the discussion, amplified in his paper, was of special interest inasmuch as it was confined to the control of subtertian malaria in war, a matter of outstanding importance. He recalled that some epidemics of subtertian malaria were uncontrollable in wars of the past, including the last war, and expressed the opinion that with the aid of synthetic drugs, in addition to quinine,

[3] HILL (T. Rowland). Subtertian Malaria in War. With Haematological Report by J. W. HOWIE.—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1942. Aug. 31. Vol. 36. No. 2. pp. 75–88. With 4 charts. [16 refs.]

it should be possible greatly to reduce the incidence of clinical subtertian malaria among troops in malarious regions. He considers that under-treatment is the greatest fault in the present-day treatment of cases of subtertian malaria in hyperendemic zones. Thirty grains of quinine a day for 5 days may bring the temperature down in an attack of subtertian malaria but in a large proportion of cases recrudescences occur a week or a fortnight later; subsequent severe anaemia and invalidism are likely to result. Exposure to strain and fatigue increase the predisposition to recrudescences. Immediate and thorough treatment should be commenced directly the clinical diagnosis is made; this should not be delayed till a positive blood-film report confirms the diagnosis. He recommends for the treatment of the acute attack 0·1 gm. [printed wrongly as 0·01 gm. on p. 73, but corrected to 0·1 gm. on p. 80] of atebrin and 10 grains of quinine three times a day for 7 days. No toxic signs or symptoms have been noted in 200 cases so treated. In severe cases these doses may be increased with advantage. During the next two days 10 grains of quinine hydrochloride are given three times a day. During the following 5 days 0·01 gm. of plasmoquine and 10 grains of quinine hydrochloride are given three times a day. The plasmoquine part of the treatment might be considered optional, perhaps, for cases treated in the field, but should be compulsory in hospital. The intravenous administration of quinine is not a dangerous undertaking; indeed it is essential at times if lives are to be saved. Medical officers should always be prepared to use it in the field. Ten grains in 10 cc. of water have very often been given intravenously; in no case have toxic effects resulted. Intramuscular injections of quinine should never be given. Extremely good results have also been obtained by the intravenous administration of soluble atebrin in doses of 0·3 gm. For anti-relapse treatment a dose of 5 grains of quinine a day, which may suffice for the needs of a civilian, is wholly inadequate for those undergoing excessive strain. Even 15 grains a day, which can safely be administered, will not always prevent clinical attacks. It is preferable to give one tablet, 0·1 gm., of atebrin every day. Prolonged trial has shown the efficacy and harmlessness of this; recrudescences are virtually abolished. Yellowing of the skin is of no consequence among troops in war. Statements regarding the toxicity of atebrin have been much exaggerated. For the rest, the use of veils and gloves, the killing of adult mosquitoes with paraffin and paraffin sprays, the use of mosquito nets by slowly moving troops, and the education and training of soldiers in anti-malarial measures, all help to reduce malaria prevalence. The haematological investigations reported by Howie show the beneficial effects of the treatment outlined above.

Manson-Bahr does not consider the intravenous or intramuscular administration of atebrin as effective as quinine administered in like manner. Intramuscular injections of quinine can be of very great value. He agreed with Rowland Hill that relapses do not occur in persons with atebrin-tinted skins.

Dove,[4] writing with the experience of 14 years in the American tropics, describes the treatment used during a period of two years in the medical care of 30,000 persons, nearly all of whom had malaria. Most of the work on which they were engaged was of a temporary nature and in unsanitated areas. Patients when cured did not return

[4] Dove (Wendell S.). The Treatment of Malaria.—*Amer. Jl. Trop. Med.* 1942. May. Vol. 22. No. 3. pp. 227–234.

to work but the labourer was unable to return home unless completely cured. Treatment was thorough. The treatment of malaria is not discussed according to the type of malaria present. Dove is of the opinion that treatment should be determined solely by the symptomatic indications presented and by the urgency of such symptoms, and not by the form of malaria. The *minimum* treatment prescribed for a case of malaria lasts about four weeks and consists of :—

(1) Atebrin, grains 1½, thrice daily, during febrile period and 4 days thereafter.
(2) Quinine, grains 5, 4 times a day for 7 days.
(3) Atebrin, grains 1½, thrice daily for 5 days.
(4) Quinine, grains 5, thrice daily for 5 days.
(5) Quinine, grains 5, and plasmoquine grain 1/6, thrice daily for 5 days.

In chronic, frequently relapsing cases periods 3, 4 and 5 of the treatment are repeated after a ten days' interval. Should the atebrin give rise to toxic symptoms, longer intervals between its administration are allowed, 15 grains of quinine being given while waiting. The author, however, has never seen a patient intolerant of atebrin though quinine idiosyncrasy does exist. In neglected cases harbouring many gametocytes plasmoquine is combined with quinine in the second period of treatment.

In the few cases in which the initial treatment by mouth is impossible, injections are given. This is only necessary in the first period of treatment and only atebrin is used for injection. The author considers that there are no indications for the injection of quinine either by the intramuscular or intravenous routes. Even with every care intramuscular quinine may cause sterile abscesses. Such industrial cases have been awarded 6 months' total temporary disability and subsequent permanent 20 per cent. partial disability, a fact which sufficiently explains the author's disapproval of intramuscular quinine injections. Atebrin if injected with sterile precautions does not produce abscesses and is not painful ; moreover, there is no evidence that atebrin will not do all that quinine can in the treatment of malaria. Disapproval of intravenous injection of both drugs is expressed. Intramuscular injections of atebrin are given (4½ grains of atebrin in 7½ cc. of distilled water, repeated after 12 hours and thereafter at 24-hour intervals till oral medication is possible) in the following circumstances :—if the patient be unable to swallow, or to retain or absorb the drug ; if there be hyperpyrexia with temperature above 104°F. ; if there be a localization of symptoms indicating a concentration of infection in important organs or systems of the body ; if an overwhelming infection be present ; or if the fever has failed to respond to oral administration. Subcutaneous injections of plasmoquine 1/3 grain are given to chronic malaria patients with severe symptoms, whose gastro-intestinal tract is not functioning normally and in whose blood are numerous gametocytes, notably those of *P. falciparum*.

In addition to describing the specific treatment of malaria the author deals well with the non-specific or symptomatic treatment of the many complications that may arise in the course of malaria. In blackwater fever specific treatment is often contraindicated until convalescence is established ; large amounts of saline-glucose are given to dilute and wash out the decomposition products of the red cell destruction.

Chemical prophylaxis against malaria is feasible but the author considers it to be justifiable only in temporary unsanitated localities, while waiting for the establishment of mosquito control, which is the

only safe prophylactic measure. Three grains of atebrin twice a week had a certain amount of success but there were cases of fever of undetermined origin in a group so protected, which were amenable to antimalarial treatment. Atebrin, having been frequently reported as a neurotoxin, should not be used over a long period of time even in small doses [but compare Hill above]. The chemical prophylaxis preferred by the author consists of the administration of 5 grains of quinine twice a day for an indefinite period. If there be any sign of intolerance, weekly rest periods are allowed, during which 3 grains of atebrin are given three times.

Not so long ago nearly every publication dealing with the relative value of quinine and atebrin, respectively, in the treatment and clinical prophylaxus of malaria, contained some statement regarding the marked superiority of quinine for mass prophylaxis. It was nearly always insisted that atebrin should in no circumstances be given without medical supervision. Recently much less has been heard of the toxicity of atebrin. BRYANT [this *Bulletin*, Vol. 39, p. 666], in the treatment of the acute attack of subtertian malaria, gives doses of atebrin two to three times greater than the "safe" doses usually recommended, and does so with impunity and profit. He considers that the simultaneous administration of sugar, in the form of very sweet hot tea, in quantity, diminishes such risk as there may be of toxic symptoms. ROSE [this *Bulletin*, Vol. 39, p. 396] tells us that the daily administration of 0·06 gm. of atebrin was the method of clinical prophylaxis used in the present war by the German army in their Balkan campaign. It was considered that larger doses once or twice a week were more likely to be overlooked than the daily small dose. Some men, and some doctors, insisted that the drug thus given had a cathartic action, but Rose was probably right in his contention that intestinal disturbances in Balkan campaigns are likely to occur, atebrin or no, and that diarrhoea is not commonly produced by much larger doses. Finally, Rowland Hill, as we have seen above, has had experience of the efficiency and harmlessness of the prolonged daily administration of 0·1 gm. of atebrin.

One may find in all this ample justification for the administration of atebrin daily, in a dose not exceeding 0·1 gm., to all susceptible troops exposed to the stress and strain of war in countries where subtertian malaria is prevalent. This measure indeed would seem to be desirable even if supplies of quinine be adequate for all military needs. Troops are at nearly all times subjected to adequate medical supervision.

[A recent account of German practice in the treatment of malaria is contained in the paper by MEYTHALER abstracted below.]

MALARIA.

MEYTHALER (F.). Die Therapie der Malaria. [**The Treatment of Malaria.**]—*Muench. Med. Woch.* 1942. Sept. 18.. Vol. 89. No. 38. pp. 812–818. With 12 charts.

This paper is of exceptional interest at the present time: the methods of treatment described have been tried on a large scale in conditions of war and presumably also of restricted supplies of quinine.

Complete success is claimed from short courses of treatment with very moderate doses of the drugs employed, but there is no suggestion that the need for economy had any influence in the choice of the system adopted.

A simple routine treatment is said to be suitable for all cases of benign tertian and quartan infections, and also for all cases of subtertian infection that comply with the following conditions :—(1) the treatment must be started within two days of the onset, (2) the infection must be known to be due to a strain of low virulence, (3) the parasites must be relatively few in number, and (4) the patient must be kept under close observation because of the possibility of a poor response or the development of indications that the infection is of a more severe type than it appeared to be.

Atebrin [mepacrine] is given thrice daily for seven days in doses of 0·1 gramme each ; two days' rest from drugs is allowed and then plasmoquine [pamaquin] is given thrice daily after food, for 3 days, in doses of 0·01 gramme each.

In all cases of subtertian infection which do not comply with the above conditions the dose of atebrin is doubled till the temperature becomes normal or atebrin musonate is given by the intramuscular route in daily doses of 0·3 gramme, dissolved in 10 cc. distilled water till the temperature becomes normal when the routine treatment can be followed for the rest of the course. In really severe attacks the author's practice is to give 0·3 gramme atebrin musonate by the intramuscular route at once ; he repeats the dose in 10 hours and then continues the injections in fractional doses to the equivalent of 0·3 gramme twice daily till the temperature is controlled.

The intramuscular route is claimed to be the method of choice in all severe cases in which the circulation is not enfeebled to such a degree as to prevent prompt absorption of the drug.

In the severest or " pernicious " attacks quinine-urethane is given intravenously in addition to atebrin musonate, which in such cases may also be given intravenously, dissolved in 20 cc. of a 10 per cent. solution of glucose, and injected *very slowly*. The dose of quinine urethane is 2 cc. (containing 0·5 gramme quinine hydrochloride) ; this is given once daily or even twice daily, and if there is no sign of circulatory failure it may be given by the intramuscular route without fear of tissue necrosis, which is said to result only from the use of the " insoluble " salts of quinine.

There may be a fall in the blood pressure to the extent of about 15 to 20 mm. Hg. after an intravenous injection of atebrin musonate, even when four minutes are spent in giving the injection, so it is suggested that repeated fractional doses may be preferable. Nevertheless in 1,500 intravenous injections no serious trouble has been experienced, and some German clinicians prefer to give intravenous injections for the first few days in all cases of subtertian infection.

The condition of the circulatory system needs special attention : strophanthin and glucose are strongly recommended. Lumbar puncture is needed in cerebral attacks. Blood transfusions, up to a maximum of 700 cc., were often given ; the indirect method is advocated, and great care must be taken in the choice of a donor, especially in malarious localities.

The author claims that the relapse rate in benign tertian infections did not exceed 20 per cent. He attributed many of the recrudescences in subtertian cases to insufficient treatment.

Extensive trials were made of " provocation " tests for the purpose of finding whether the patient was fit for return to duty in conditions in which proper treatment would not be available for relapses. The tests were made two or three weeks after the end of the courses of treatment and consisted in the subcutaneous injection of 0·5 mgm. adrenalin, followed by blood examination and careful observation of the rectal temperature. The proportion of positive results was not large, but no really harmful results were observed. The method is considered suitable only for young patients who are treated in hospitals in which skilled observation is possible. Negative findings are not to be regarded as proof of cure.

[It might be suggested that a second course of treatment, perhaps rather shorter than the first, and started about a fortnight later, would be a simpler and better method of dealing with the situation. The author regards atebrin as a perfectly safe drug with practically no contraindications, so that it can be used freely as a means of diagnosis in doubtful cases, but it is interesting to note that quinine is used in the very worst attacks. This suggests that it may be desirable to give a few initial doses of quinine in all cases of subtertian infection, either alone or in combination with atebrin, even when strict economy must be observed in the use of quinine.] *John W. D. Megaw.*

Russell (Paul F.). & Menon (M. Kumara). **A Malario-Economic Survey in Rural South India.**—*Indian Med. Gaz.* 1942. Mar. Vol. 77. No. 3. pp. 167–180. With 2 figs. [13 refs.]

This paper records the results of a house-to-house survey of five villages in the Tanjore District of Madras. Three of these were in the Pattukkottai *taluk*, where the Cauvery-Mettur canal irrigation scheme, which came into operation in 1933, was responsible for *A. culicifacies*-carried malaria in an area that had till then been free of the disease ; two of the villages were in the Papanasam *taluk*. The Papanasam *taluk* is in the Cauvery delta and here irrigation by canal direct from the Cauvery River has been practised for centuries ; it is completely free of endemic malaria. Those who have had practical experience of the difficulties attendant upon any attempt to carry out surveys of this kind in Indian conditions, and of the difficulties of assessing the value and significance of the information so collected, will read this report with interest not untinged with admiration.

The main purpose of the survey was to arrive at an estimation of the economic burden that malaria imposes on an Indian rural community and to find out whether such a community could provide from its own resources funds sufficient to control the disease. It is not possible in small space to summarize the information collected. In two malarious villages, days lost owing to fevers averaged 4·9 and 10·5 per head per annum ; payments for malarial fevers averaged **Re.* 1–6–0 and *Rs.* 2–8–0 and wages lost *Re.* 0–12–0 and *Re.* 1–6–0 per head per annum. Writing of the cost of illness in one community the authors state " The largest items of expense were for travel, offerings, and temple sacrifices of fowls, sheep or goats. Temple and church candles cost *Re.* 0–0–9 each and generally one to three will be purchased during such an illness as an attack of malaria."

It is concluded that the direct financial loss due to malaria is considerably more than the amount it would cost to control the disease.

* A rupee equals 1*s.* 6*d.* ; there are 16 annas in a rupee and 12 pies in an anna.

Russell and Knipe [this *Bulletin*, 1941, Vol. 38, p. 645] found that malaria could be controlled in Kasangadu, one of the villages included in the survey, by spray-killing adult mosquitoes, at a cost of *Rc.* 0–7–2 per head per annum, and that by using Indian grown Pyrethrum the cost might be reduced to *Re.* 0–4–7 per head. Bearing in mind, however, the very small residue of income left after paying taxes and interest on debts, the imposition of a special malaria tax is not possible. As irrigation water is chiefly responsible for the malaria in this region the authors consider that it would be reasonable for a percentage of the irrigation tax to be set aside for malaria control. Once more the authors plead for more effective co-operation between agriculture, public works and the health departments.

Norman White.

de Zulueta (Julián). *Plasmodium ovale* en Colombia. [*P. ovale* **in Colombia.**]—*Rev. Facul. de Med.*. Bogota. 1942. Jan. Vol. 10. No. 7. pp. 487–502. With 2 plates. [Bibliography.] English summary (8 lines).

The author describes a case of locally acquired malaria in Colombia. The blood contained numerous parasites which morphologically were indistinguishable from *P. ovale*. In addition there were numerous typical *P. vivax* parasites and a variety of forms partaking of the characteristics of both. Hitherto *P. ovale* has not been reported from Colombia. The author's observations lead him to believe that *P. ovale* is but a variety of *P. vivax*. The paper contains references to a very large number of the papers concerning *P. ovale* that have been published since Stephens's discovery of the species in 1922. The bibliography contains 31 references. *Norman White.*

du Toit (Maria L.). **A Case of** *Plasmodium ovale* **Infection.**—*South African Med. Jl.* 1942. May 9. Vol. 16. No. 9. p. 182. With 1 fig.

The case occurred in Swaziland, in a native woman who complained of mild attacks of fever. Drawings made of cells and parasites leave little doubt that it was a *P. ovale* infection. *C. W.*

Blacklock (D. B.) & Wilson (Carmichael). **Apparatus for the Collection of Mosquitoes in Ships, with Notes on Methods of Salivary Gland Dissection.**—*Ann. Trop. Med. & Parasit.* 1942. June 30. Vol. 36. Nos. 1 & 2. pp. 53–62. With 8 figs.

For the collection of mosquitoes on board ship the authors devised a light compact equipment consisting of a torch and a small portable box containing a rack for holding tubes. Round bottom tubes 2 inches by ½ inch, plugged with cotton wool, were found satisfactory. Mosquitoes were collected singly in these and they could generally be identified while still alive in the tube.

The paper includes a useful illustrated summary of the various methods that have been proposed for dissecting out the salivary glands of mosquitoes. The authors propose a method which is substantially

the same as that of SHUTE, though perhaps even simpler. A fine mounted needle or entomological pin and a narrow bladed scalpel (one-tenth inch or less) are employed. The mosquito is killed, freed from legs and wings and laid in a drop of saline on a slide. The neck is cut cleanly through with the knife. The blade is then placed across the thorax so that very gentle steady pressure can be applied at a point just behind the origin of the first pair of legs. The glands will then emerge from the cut end of the neck. In the absence of chloroform ordinary motor car petrol applied to the cotton wool plug (preferably with a dry plug beneath) forms a satisfactory anaesthetic.

V. B. Wigglesworth.

MACAN (T. T.). **A Key to the Anopheline Mosquitoes of the Mediterranean Region and the Lands adjoining the Red Sea and the Persian Gulf.**—*Jl. Roy. Army Med. Corps.* 1942. July. Vol. 79. No. 1. pp. 1–11. With 14 figs. & 1 map.

One or two of the locality records are erroneous.

P. A. Buxton.

WEYER (F.). Beitrag zur Stechmückenfauna von Mazedonien und Westthrazien. [**The Mosquitoes of Macedonia and Western Thrace.**]—*Deut. Trop. Ztschr.* 1942. May 15 & June 1. Vol. 46. Nos. 10 & 11. pp. 249–257 ; 284–293. With 3 figs. [Numerous refs.]

WEYER (F.). Die afrikanischen Malariaüberträger. [**The Vectors of Malaria in Africa.**]—*Deut. Trop. Ztschr.* 1941. Feb. 15. Vol. 45. No. 4. pp. 112–118.

SEN (P.). **Distribution of *Anopheles sundaicus* Rodenw. through Mechanical Means.**—*Jl. Bombay Nat. Hist. Soc.* 1941. Vol. 42. No. 3. pp. 593–598. With 1 map. [Summary taken from *Rev. Applied Entom.* Ser. B., 1942. July. Vol. 30. Pt. 7. p. 106.]

The transport of *Anopheles sundaicus*, Rdnw., to Calcutta from its breeding places in the Sunderbans by train and country boat during 1933 and 1934 is recorded. Collections made from 234 trains at the terminal railway stations in Calcutta that have connections with the outlying breeding zones included 101 individuals of *A. sundaicus*, taken in 35 trains, as well as several other species that do not transmit malaria in the area involved. Those identified were *A. hyrcanus*, Pall., *A. barbirostris*, Wulp, *A. annularis*, Wulp, *A. ramsayi*, Covell, *A. philippinensis*, Ludl., *A. vagus*, Dön., *A. subpictus*, Grassi, *A. varuna*, Iyen., *A. aconitus*, Dön., and *A. stephensi*, List. Of 3,062 country boats arriving at Calcutta after passing through areas infested with *A. sundaicus*, 136 carried a total of 142 individuals of this species under shelter of the cargo, and *A. subpictus*, *A. vagus* and *A. varuna* were also found in them. The most favourable resting places were in cargoes of rice, jute and straw. Dispersal in trains was most noticeable between June and September and by boat from July to December. It is thought that the establishment of *A. sundaicus* in the Salt Lake area to the east of Calcutta has been the outcome of importation in boats, as no trains stop near the Salt Lakes.

Rozeboom (L. E.). **Subspecific Variations among Neotropical Anopheles Mosquitoes, and their Importance in the Transmission of Malaria.**—*Amer. Jl. Trop. Med.* 1942. May. Vol. 22. No. 3. pp. 235–255. With 31 figs. on 5 plates. [74 refs.]

In this paper the author brings together present knowledge about the variations in the commoner Anopheline mosquitoes of the New World tropics. In some of these, such as *A. punctipennis*, there are well-marked differences in relation to malaria in different localities and there are differences in egg structure; but an exact correlation between the two has yet to be proved. In others, such as *A. albitarsis*, the egg structure seems to be constant but there is strong evidence of biological variations; in some regions this mosquito bites man and is a carrier, in other regions it ignores man and is harmless; the adult characters show great variation but do not seem to be correlated with the differences in habit. *A. aquasalis* (*tarsimaculatus*) again seems to comprise at least two sub-species: it is a man-biter and malaria carrier in Trinidad, ignores man and is harmless in Panama. But structural differences in egg or adult are not well defined. The author concludes that much more work is needed before definite statements can be made on the subspecies of neotropical anophelines. Such questions as the effect of environment on the morphology of eggs or on the behaviour of adults, the geographical distribution of egg types and whether these change gradually in passing from one locality to another, and the cross breeding of races laying different eggs all require detailed study. *V. B. Wigglesworth.*

Smith (Gordon E.). **The Keg Shelter as a Diurnal Resting Place of *Anopheles quadrimaculatus*. Introduction.**—*Amer. Jl. Trop. Med.* 1942. May. Vol. 22. No. 3. pp. 257–269. With 6 figs.

During five years the author has tried many methods of measuring the density and composition of the Anopheline fauna in the South Eastern United States. Light traps, various combinations of animal baited traps, earth dug-outs and box shelters of many sorts proved unsatisfactory. The most promising results were obtained with small nail kegs [apparently about ten inches in diameter] laid on their sides in the most deeply shaded spots along the shore line near the breeding places. The mosquitoes, both males and females, enter these at dawn at the first sign of light and remain there until dusk. Both *A. quadrimaculatus* and *A. punctipennis* are collected. The numbers of mosquitoes entering them increase and decrease more or less concurrently with the numbers of engorged females in barns in the same localities; they probably afford, therefore, a true measure of the local mosquito population. It is claimed that these keg shelters have advantages of uniformity and mobility which are lacking in conventional collecting stations. *V. B. Wigglesworth.*

Anderson (W. A. D.), Morrison (Dempsie B.) & Williams (E. F.), Jr. **Pathologic Changes following Injections of Ferrihemate (Hematin) in Dogs.**—*Arch. Pathology.* 1942. May. Vol. 33. No. 5. pp. 589–602. With 7 figs. [17 refs.]

In the course of a study of the solubility and the titration of haemin and ferrihaemic acid, Morrison and Williams (1941) prepared a suitable

solution of disodium ferrihaematе of *p*H 7·6. It was considered that such a well buffered solution might be of value for the relief of anaemia, and accordingly some of it was administered intravenously to a dog which had undergone severe haemorrhage, but the result was the death of the dog. This fatal result was repeatedly confirmed in apparently normal dogs without previous haemorrhage. The authors became interested in the identification of malaria pigment with ferrihaemic acid, and the question was raised whether the toxic symptoms in cases of fulminating malaria might be due to release of ferrihaemic acid.

Solutions of disodium ferrihaematе prepared from recrystallized haemin and adjusted to a *p*H of 7·6 were injected into dogs intraperitoneally, subcutaneously and intravenously. Marked reactions were induced by intravenous injections, death occurring when an injection was made too rapidly or when excessive amounts were given. In one dog, anuria developed two days after the injection and persisted for 36 hours. Two dogs which survived intravenous injection were killed on the 4th and 6th days after the injection, respectively. Intraperitoneal injections were well tolerated. After several subcutaneous injections the injected areas became so oedematous and tender that further injections were not attempted.

The results of this work are described in full ; the following summary is given :—

" Ferrihemate when injected into dogs is capable of producing acute and chronic changes in the kidneys, the reticuloendothelial system and the vascular system.

" When stored in the reticuloendothelial system, ferrihemate remains as an inert and relatively harmless substance, which the body appears to have little ability to metabolize.

" Marked vascular reactions followed injection, particularly intravenous injection, of ferrihemate. The vascular changes consisted of dilatation and congestion of small vessels, hemorrhages and thromboses. Small hemorrhages were common in the central nervous system.

" The renal lesions were similar to those which may follow marked intravascular hemolysis, as from transfusion of incompatible blood, but without as marked tubular blockage. The renal degenerative changes were probably due to vascular injury and obstruction rather than to any inherent specific toxicity of ferrihemate.

" Many of the changes following injections of ferrihemate are similar to lesions which occur in malaria and blackwater fever.

" Siderofibrotic nodules of the spleen (Gandy-Gamna bodies) resulted from deposition of ferrihemate in the spleen." [See also this *Bulletin*, 1942, Vol. 39, p. 604.] *W. Yorke.*

BELTRAN (Enrique). Infección de reticulocitos por *Plasmodium vivax*. [**Infection of Reticulocytes by** *P. vivax*.]—*Rev. Inst. Salubridad y Enfermedades Trop.* Mexico. 1941. Dec. Vol. 2. Nos. 3–4. pp. 327–345. [32 refs.] English summary.

This is a study of the preference exhibited by the merozoites of *Plasmodium vivax* for reticulocytes. The author discusses at some length the findings of the large number of workers who have dealt with this question and describes his own observations. Six cases of naturally acquired malaria, and three cases of neurosyphilis undergoing malaria therapy were studied. The blood of some of these patients was

examined but once; of others repeated examinations were made. The proportion of reticulocytes to the total number of red cells varied from 0·2 to 6·8 per cent. The percentage of ring forms of *P. vivax* found in reticulocytes varied from 12 to 80 per cent. These proportions varied greatly in different cases and also in individual cases from day to day. *Norman White.*

AZEVEDO (Alipio). Um caso curioso e ligeiras considerações em torno do impaludismo. [**An Unusual Case of Malaria and Some Slight Observations concerning that Disease.**]—*Brasil-Medico.* 1942. Feb. 14. Vol. 56. No. 7. pp. 84–85.

This short note describes the case of a woman, aged 30, suffering from malaria who, as a prelude to febrile attacks, suffered from very severe epigastric pain, simulating hepatic colic, and urticaria with intense irritation, chiefly on the arms and neck. These distressing symptoms lasted about six hours, but reappeared 24 hours after the onset of the first attack. After three such attacks the periodicity and some slight enlargement of the spleen suggested malaria, a diagnosis which was confirmed. Specific treatment effected a cure. The patient had never previously suffered from any allergic conditions, and she had taken neither quinine nor other drug that might have caused the urticaria.

The author offers some remarks concerning the inadequacy of much vaunted anti-malarial drugs, including the well-known synthetic remedies. A combination of methylene blue and quinine has given good results in his hands. He comments on the frequent association of malaria with cardiac disorders, chiefly cardiac arhythmia. In the malarious regions of the Province of São Paulo the association of mega-oesophagus and megacolon with malaria infection is sufficiently constant to justify the assumption that malaria is responsible for these disorders. *Norman White.*

SANDERS (J. P.). **Ten Years' Experience treating Malaria by the Short Course Method.**—*New Orleans Med. & Surg. Jl.* 1942. Apr. Vol. 94. No. 10. pp. 465–469. [12 refs.]

The author has previously published several papers recording very good results in the treatment of malaria by short courses of quinidine. Until 1933 his short course consisted of four single daily 10 grain doses of quinidine; he then increased the course of treatment to five days, giving two doses of 10 grains each on the first two days, and single doses of 10 grains on the remaining three days, 70 grains in all. In the present communication he summarizes some of the results of his experience in the treatment of malaria during the period 1930 to 1940. Of a total of 3,859 [wrongly printed as 3,656] cases, 2,215 were treated with quinidine, 1,449 with quinine, 108 with atebrin, and 87 with other drugs. The author states that there is a strange similarity between all the anti-malarial drugs in that they "cure" from 65 to 75 per cent. of cases. Idiosyncrasy is about as common with quinidine as with quinine; there were six cases among the 2,215 patients treated, the reaction varying from a very mild to a moderately severe urticaria. Patients intolerant of quinidine may be able to

take quinine, and *vice versa*. Quinidine does not cause uterine contractions and so can be given safely in all stages of pregnancy ; neither does it cause tinnitus. Patients who develop haematuria during the administration of quinine tolerate quinidine, though the latter drug may very exceptionally give rise to haematuria. *Norman White.*

BLACKLOCK (D. B.). **The Prevention of Mosquito-borne Diseases in Tropical and Sub-Tropical Towns.**—*Ann. Trop. Med. & Parasit.* 1942. June 30. Vol. 36. Nos. 1 & 2. pp. 63–74.

This paper describes the methods at present employed in Freetown, Sierra Leone, to search out and eliminate mosquito breeding places, and the training, qualifications and duties of the staff engaged on this important work. It is pointed out that the number of breeding places discovered is very small in comparison with the high malaria endemicity of the town. The author makes suggestions as to how the work might be more effectively and systematically carried out than at present, and at less cost. A complete scheme is elaborated in detail ; this does not lend itself to summary. Health officers of tropical towns will find in this paper suggestions, concerning the details of mosquito control work, which are likely to be of great assistance. *Norman White.*

WATSON (Malcolm). **Some Emergency Anti-Malarial Measures.**—7 pp. With 1 plan & 8 figs. 1942. May. London : Ross Institute Indust. Adv. Cttee.

In view of the loss of oil supplies in the Far East, and of the necessity for conserving oil and reducing transport of oil in war time, it is necessary to consider what methods of malaria control can be employed which do not involve the use of oil. While emphasizing that under certain conditions oil is imperative, the author describes other methods which may be useful, especially the methods of sluicing or flushing. Considerable detail is given and there are several illustrations. Reference is also made to the value of rotting vegetation, faggot drains and anti-malarial hedges. *C. W.*

SCHARFF (J. W.) & TWEEDIE (M. W. F.). **Malaria and the Mud Lobster.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1942. June 30. Vol. 36. No. 1. pp. 41–44. With 2 figs.

The mud lobster, *Thalassina anomala*, a crustacean some eight inches in length, resembling a crayfish, lives in the soft mud underlying the superficial hard soil in brackish swampy areas of Malaya. It tunnels upwards to the surface and forms "crab holes," a misnomer arising from the fact that crabs use the burrows as a refuge. These mud-holes cause much damage by undermining tidal bunds, and may also provide breeding places for mosquitoes.

A description is given of experiments carried out on the east coast of Singapore Island with the object of finding a cheap and reliable method of destroying *Thalassina anomala*, and of preventing its burrowing in tidal bunds. A quarter to half a gallon of lime and water, 5 pounds of lime to 3 gallons of water, poured into the hole, which is then rammed with sand, destroys the lobster. This method conscientiously applied can free an area ; periodical inspection and treatment of new holes round the margin of the area can eliminate the mud lobster.

A layer of the fibrous material formed on the trunks of coconut palms by the withering of the leaf bases, soaked in coal tar, incorporated in a tidal bund, one foot from the top, will prevent the mud lobster from burrowing up through the bund. *Norman White.*

Fulton (J. D.). **Attempts to prepare in Fowls a Strain of *Plasmodium gallinaceum* Resistant to Plasmoquine.**—*Ann. Trop. Med. & Parasit.* 1942. June 30. Vol. 36. Nos. 1 & 2. pp. 75–81. [12 refs.]

The author describes an attempt to produce a strain of *Plasmodium gallinaceum* resistant to plasmoquine by keeping infected fowls, during 33 passages over a period of 15½ months, under the influence of plasmoquine administered orally. At the end of this period the strain which had been kept exposed to plasmoquine was no more resistant to the drug than was the original unexposed strain. The question arose as to whether this failure to become resistant could in any way be correlated with the presence of exoerythrocytic schizonts, which various workers have shown to be unaffected by quinine, atebrin or plasmoquine. In the present investigation the author examined a number of treated and untreated birds and found that there was no difference in the frequence of occurrence of exoerythrocytic schizonts. It would seem, therefore, that the resistance of the exoerythrocytic schizonts to drug treatment may account for the failure to promote a drug-resistance.
C. M. Wenyon.

TRYPANOSOMIASIS.

Congo Belge : Rapport sur l'Hygiène Publique au Congo Belge pendant l'Année 1940 [van Hoof (L.)]. pp. 24–37. With 1 folding map.—Trypanosomiase. [**Trypanosomiasis.**]

For more than 15 years each big endemic area has been supervised by a branch of the Medical Services, assisted by auxiliary services of missionaries or private enterprises. Although circumstances forced these medical missions to concern themselves with all types of maladies and health services, they dealt primarily with sleeping sickness. As the principal attack was to destroy the human reservoir of infection by drug control, as complete a survey of the natives as possible was required, and owing to difficulties between the medical and administrative surveys, it was necessary for a survey to be made by medical personnel dealing with all affections susceptible of treatment by a rural medical service. It is obvious that the reduction in personnel due to the war made this difficult and the survey was therefore provisionally suspended in certain sectors considered as little affected or practically cleared. Nevertheless the whole of the endemic area was inspected, special attention being given to areas of high endemicity, certain of which were visited at least four times during the year. Close on 5 million natives were examined, 11,837 new cases of trypanosomiasis were discovered and there were also 35,189 old cases under control. There were 4 cases in Europeans, 1 in the Province of Léopoldville and 3 in the Province of Costermansville.

While the index of new infections decreased in general in the Colony, the frequency of drug resistant cases increased. This is explained in

part by the persistence of certain foci despite all efforts ; in such areas control with Belganyl (Bayer 205) leads to a massive but not complete reduction in the endemic index.

Researches on methods of diagnosis and treatment, biological variation and transmissibility of trypanosomes, identification of species, synergism of remedies, clinical aspects of the disease and its complications in man and animals, have been carried out during the year. A new rural hygiene ordinance will facilitate the task of the medical services in the control of tsetse flies.

Province of Léopoldville : The whole province shows some increase for the year ; 1,584,846 persons were examined and 5,671 new cases were discovered, giving an index of new infection of 0·36 per cent. The principal foci are along the Congo River from Léopoldville to Kwa, along the Kasai and in the Kwango to the Middle Wamba and to the east of the Kwili. In the last two areas some recrudescence was observed but that of Chenal on the contrary appeared to have been suppressed by extensive use of Bayer 205 for prophylaxis. A violent recrudescence on the Portuguese border between Kwango and Wamba is reported.

Province of Lusambo : In this vast endemic area a notable diminution in the index of new infections for the year was obtained, owing to intensification of measures in areas shown to be most menaced in the previous years' surveys. In 1940, examinations of 935,792 persons were made and 2,163 new cases were discovered, giving an index of new infection of 0·23 per cent. The tremendous loss of ground and the danger of serious recrudescence following even temporary relaxation of effort in zones incompletely cleared of infection is stressed.

Province of Coquilhatville : Of 897,816 persons examined 1,429 were found to be newly infected, giving an index of new infection of 0·16 per cent. Medical control has accelerated the natural regression of the disease in the South (Tshuapa) and combated with success its extension in the North. In the interior and along the numerous tributaries of the Congo the situation improves constantly, but the basin of the river itself, with its great islands, inhabited or frequented by fishermen, constitutes an active and dangerous area resistant to control owing chiefly to the movement of the population and topographical difficulties. Commerce and the constant traffic of the fishermen favour the extension not only of sleeping sickness, but also other diseases.

Province of Stanleyville : The condition is in general stationary, special attention having been given to the active foci found in 1939. Among 552,697 persons examined 192 new infections were found, giving an index of new infection of 0·039 per cent. The region of Lake Albert, where 10 per cent. of a population of almost 100,000 were infected in 1923, remains completely clear, a result now maintained for more than 5 years.

Province of Costermansville : Among 518,210 persons examined 1,352 new infections were found, giving an index of new infection of 0·26 per cent. On Lake Edward the favourable results now permit the reopening of fisheries and the reoccupation of pastures, under medical supervision and with the aid of drug control. In the Ruzizi valley and on Lake Tanganyika, where movement of natives is now freely permitted, the endemic is still receding. In Maniema the large areas of infection remain confined along the Lualaba and the Kongolo-Kindu railway.

Province of Elisabethville: Among 350,733 persons examined 1,030 new infections were found, giving an index of new infection of 0·29 per cent. An increase in endemicity is recorded on the Lualaba, principally at Kongolo, Mukula-Kulu. On the other hand improvement is reported on Lake Tanganyika. The Kongolo focus is a prolongation of the large endemic area from the Lualaba to Maniema, and is favoured by the floating population and the recruitment of native labour. On Lake Tanganyika endemicity is maintained in fishermen and the prophylactic use of Bayer 205 in professional fishermen is suggested.

F. Murgatroyd.

Jack (R. W.). **Further Studies in the Physiology and Behaviour of *Glossina morsitans*, Westw.**—*Mem. Dept. Agric. S. Rhodesia.* Salisbury. 1941. No. 3. [4] + 56 pp. With 6 charts. [Summary taken from *Rev. Applied Entom.* Ser. B. 1942. Aug. Vol. 30. Pt. 8. pp. 115–117.]

This paper comprises records collected on the eco-climate of various situations in the Chipane area of Southern Rhodesia in which *Glossina morsitans*, Westw., occurs and the results of observations on the bionomics of the fly in the field, made by W. L. Williams. The observations were planned as an extension of the author's laboratory research. The eco-climatic records were collected from July to November 1938 in six situations, of which two were in evergreen river forest, two in savannah forest, one in open country at an altitude of 3,000 ft., and one in open country at an altitude of 1,400 ft. in the Zambezi valley. The findings at this last-named station show that the evaporation rate is very high during the latter part of the dry season. There were nine days on which the temperature in the screen rose to 104°F. or over, whilst on four days it rose to 106°F. or over.

It is considered evident that in many localities, cooler refuges than those afforded by simple shade must be essential for *G. morsitans* during the latter half of the dry season, and it is suspected that in exceptionally hot periods, the fly might suffer a severe setback. Records obtained at the other stations showed that flies in the shelter of certain kinds of riverine forest are exposed to a much lower evaporation rate than is apparent from standard meteorological records.

The following is based on the author's summary of data obtained in the course of the observations on bionomics. They showed a great variation in the weight of blood ingested at a meal by flies of the same sex, whether they were wild flies or unfed reared ones offered food on the day after emergence. There was also great variation of weight among flies caught at the same interval after feeding, and it is considered that the weight of a fly has relatively little value in indicating the period that has elapsed since its last meal. A meal caused a marked depression in the water ratio (percentage of water to wet weight less fat) on the day after feeding, followed by a rise and then a gradual decline if another meal was not obtained. The weight of fat in wild flies caught at the same interval after a meal was very variable. The percentage of wild flies recaptured after being fed and released was much higher in both sexes than the percentage of bred flies recaptured after being fed once on the day after emergence and then released. Flies taken on the higher veldt had, on an average, a higher water ratio than flies taken in the Zambezi Valley. The males caught in the valley had a lower mean fat content than those taken on

the higher veldt, although the contact between game and fly is judged to have been greater in the valley. When young flies were excluded, the water ratio in females caught on moving man was lower than that in the males. Following a week of excessively hot, dry weather in the valley, flies of both sexes taken on cars and on man had a very low water ratio, and were very reluctant to leave the shade. Unfed flies with a low fat content (under 0·5 mg.) had a significantly lower average water ratio than unfed flies with a higher fat content, particularly at the higher altitude. Flies with a water ratio of 73 per cent. and over were shown to be either unfed flies not long emerged, flies that imbibed blood at the time of capture, or flies with a considerable fat reserve. Fed males with a fat content of under 1 mg. had, on an average, significantly lower water ratios than those with a higher fat content. Individual analyses of old flies of both sexes caught on the higher veldt and having a water ratio of 67·2 per cent. or less and comparison with controls with a similar weight of non-fatty solids showed that the flies were definitely short of water, the fat content being also generally low, and it is concluded that flies do lose water in excess of replacement in the field. It is deduced that at the time of year concerned, flies maintain their water content by metabolism of fat as long as a considerable reserve of this remains, but that loss of water becomes more rapid than production as the fat reserve diminishes. Data on the accumulation of non-fatty solids are presented. The nature of these solids requires investigation. They outlast the fat during starvation.

The results are also given of observations on the percentages of young males and young females to the total catch of the same sex on different attractants.

A table is given showing the weights of wild pupae collected weekly in the field, the percentage parasitised (average 4·1) and the percentage that produced adults (average 77). Whilst the lowest percentage of emergence (56) was obtained from pupae collected at the end of the hottest and driest week of the period, it is considered questionable whether this was due to the conditions to which the pupae had been exposed. Comparison between the weights of pupae bred in the laboratory and of others collected in nature suggest that by proper feeding of the females at a suitable temperature (*e.g.*, 24°C. [75·2°F.]), pupae can be obtained in the laboratory that are as heavy as the average wild ones. A correction to a previous paper, which has already been noticed, is appended.

ZUMPT (F.). Die Tsetsefliegen und ihre Bekämpfung. [**Tsetse Flies and their Control.**]—*Deut. Trop. Ztschr.* 1941. Feb. 15. Vol. 45. No. 4. pp. 118–124. With 3 figs. (1 map).

KUNERT (H.). Die Einwirkung von intravenösen und peroralen Gaben von Bayer 205 auf *Trypanosoma rhodesiense* im Liquor cerebrospinalis. [**The Action of Bayer 205 Given Intravenously and by the Mouth in *T. rhodesiense* Infection of the Cerebrospinal Fluid.**]—*Zent. f. Bakt.* I. Abt. Orig. 1942. Feb. 16. Vol. 148. No. 6. pp. 279–289. [12 refs.]

The remarkable action of Bayer 205 in first stage cases of sleeping sickness is common knowledge, and there is also little doubt that the drug can certainly cure cases in which the cerebrospinal fluid has only recently become involved. The varying number of trypanosomes in

the spinal fluid in late cases makes it difficult to form an estimate of the action of Bayer 205 in such patients. The parasites may disappear from the spinal fluid for a longer or shorter period after treatment, but this may also happen in untreated cases. The fact that late stage cases may not be cured either with Bayer 205, or with a combination of this drug and other compounds, raises the question whether the concentration of the drug reaching the cerebrospinal fluid is not very much less than in the blood and thus insufficient to exert a trypanocidal action.

The author has attempted, in a series of cases of sleeping sickness in Tanganyika, to raise the concentration of the drug in the blood by intravenous injection at short intervals of 3·0 gm. of Bayer 205, hoping thereby so to increase the concentration in the cerebrospinal fluid as to cure the disease. Details are given of 11 cases treated; some of them received a preliminary dose of 3·0 gm. by the mouth, and then doses of 3·0 gm. were given intravenously at approximately 5-day intervals.

Another series of experiments was undertaken to ascertain the concentration of Bayer 205 in the serum and in the cerebrospinal fluid of sleeping sickness patients on the 1st, 2nd, 3rd, 4th and 5th days after a 3-gm. dose intravenously and by the mouth, respectively. It was found that with either method of administration the drug reached the spinal fluid. The content in the serum was two or three times as great as in the spinal fluid after intravenous injection and about 4 times as great after oral administration. The figures obtained are given in tables.

The following are the conclusions :—

The unsuccessful treatment of late stage cases of *T. rhodesiense* sleeping sickness is reported. Large doses of Bayer 205 (3 doses of 3·0 gm. at 5-day intervals) produced only a temporary action on the trypanosomes. Apparently the relapse in the cerebrospinal fluid must be due to drug-resistant trypanosomes. Decrease in the cell count of the cerebrospinal fluid, or the absence of trypanosomes, does not imply cure; on the contrary, however, an increase in cell count is always a sign of relapse. The trypanosomes usually reappear some months later in the spinal fluid. A satisfactory demonstration that the fluid is free from trypanosomes can be made only by centrifugation and examination of the deposit.

Bayer 205, when given in a 3-gm. dose by the mouth does not make the spinal fluid negative earlier than 48 hours after administration and may not do so for 72 hours. After 6 days the parasites are again visible in the fluid. Chemical analysis confirms the clinical observations that Bayer 205 reaches the spinal fluid after oral administration. The place from which the trypanosomes reach the spinal fluid is considered and the conclusion is reached that they do so from the tissue of the choroidal plexus. *W. Yorke.*

Friedheim (Ernst A. H.). L'acide triazine-arsinique dans le traitement de la maladie du sommeil. [**Triazine-Arsinic Acid in the Treatment of Sleeping Sickness.**]—*Ann. Inst. Pasteur.* 1940. Aug. Vol. 65. No. 2. pp. 108–118.

This paper is the same as that abstracted in this *Bulletin*, 1941, Vol. 38, p. 634. *C. W.*

OTÁLORA (Benjamin). Enfermedades de Chagas en Colombia. [**Chagas's Disease in Colombia.**]—*Med. y Cirugia.* Bogota. 1942. Feb. Vol. 6. No. 6. pp. 253–5, 257–63, 265–8.

This paper, giving an account of the first cases of Chagas's disease found in Colombia, was read before the Society of Pediatrics. Knowledge of the existence of the disease in Colombia dates from Dr. PIEDRAHITA's finding of infected *Rhodnius prolixus* in 1929. The author himself has examined children from various districts where this insect is common and though blood smears did not reveal trypanosomes, xenodiagnostic methods proved 13 to be positive out of 512 (2·5 per cent.). Those infected belonged to the poorer families living in grass-roofed huts infested with Rhodnius. For epidemiological purposes the author urges a more extensive examination of the local Reduviidae and of the animals, to find out which are reservoirs of the trypanosome.

H. Harold Scott.

MAZZA (Salvador). Die Behandlung der akuten Chagas-Krankheit mit 7602 (Ac) "Bayer." [**Treatment of Acute Cases of Chagas's Disease with 7602 (Ac) "Bayer."**]—*Deut. Trop. Ztschr.* 1941. Oct. 1. Vol. 45. No. 19. pp. 577–590. With 3 figs. [17 refs.]

This is an interesting account by Professor Mazza of the results of his work with Bayer 7602 on Chagas's disease. It does not, it is true, contain much that is new or is not already known to readers of this *Bulletin*, but it constitutes an excellent summary of experimental work with the drug and the subsequent application of it in human cases of the disease. The author starts with a note on the history of the drug. In 1937 [so states the author, and this is the date of the reference given in the bibliography, but later on he says that "in 1936 he obtained the drug in a new form, a 3 per cent. solution for testing"] H. JENSCH working on substitution products in the quinolin ring prepared a compound by union of two aminomethyl-quinolin groups with one dialylmalonyl group which seemed to have a specific action on *T. cruzi*, though inactive on other trypanosomes. It was designated "Bayer 7602" (Ac) and it was sent to Prof. Mazza for purposes of research.

He started his tests with infected dogs. Details of five are given; the upshot of them is that, injected in doses of 30–50 mgm. per kgm. body weight 7602 caused the trypanosomes to disappear from the circulating blood. "The action is not a direct but an indirect one in the tissues, destroying the forms which pass out into the circulation, and so preventing accumulation of the parasite in other organs." He next made trial of the drug in naturally infected armadillos (*Chaetophractus vellerosus pannosus*) and then in mice.

He now felt justified in trying the effect in human cases of the disease and for this purpose used a 3 per cent. solution for intramuscular injection. A child of 3½ months, weighing 6·5 kgm., was given 0·25, 0·5, and three injections of 1·0 cc., altogether 3·75 cc., or 18 mgm. per kgm body weight. After the second dose (*i.e.* after 22·5 mgm., or only 3 mgm. per kgm.) no trypanosomes could be seen in a blood smear, whereas before the beginning of treatment, 300 could be found in a thick drop. Three months later, however, xenodiagnostic methods showed that infection was still present, 30 per cent. of *Triatoma infestans* larvae being infected. Obviously the dose used had been insufficient.

Professor Mazza then treated 37 patients in the first stage of the acute disease, keeping the dose low enough to avoid albuminuria or other signs of renal mischief, and then 25 more, using the 3 per cent. solution ready prepared. The trypanosomes disappeared from the circulation within 48 hours. Intravenous injection caused sweating and praecordial pain and distress.

A new form of the drug was then made available—ampoules containing 0·15 gm. of the dried substance for making a 3 per cent. solution by dissolving in 5 cc. With this, pain at the site of injection was less. The dosage of this was, at least, 30 mgm. per kgm. body-weight, but three or even four times this was tolerated. Some details of 17 cases are given, in which the doses varied from under 30 mgm. to 120 mgm. per kgm. The higher doses were needed for patients with symptoms of meningo-encephalitis. The doses must be given without rest periods unless albuminuria is produced.

Antimonials, such as neo- or solustibosan, given with a view to eliminating leishmanial forms in the tissues, possess no advantage over 7602 in proper doses, whether given before or after the 7602. By proper or "adequate" doses the author means "100–120 mgm. per kgm. for small children, 60–90 mgm. for older children, and 30–60 mgm. for adults (per kgm. body weight)." These refer to "acute stages," first or second. For those in the third stage, with myocardial and nervous symptoms, the author is not dealing in this article.

H. Harold Scott.

FEVERS OF THE TYPHUS GROUP.

Medical Research Council. *Monthly Bull. Emergency Public Health Lab. Service.* 1942. Aug. pp. 6–9. (Not for sale.)—**The Weil-Felix Reaction of "Normal" Sera.**

This report is of great interest and importance.

The agglutinin content of 100 "normal" sera for *Proteus OX19*, *OX2* and *OXK* was tested. Twenty-five of the sera were from healthy Italian prisoners of war, the rest were from persons whose blood had been sent for examination in connexion with syphilis, gonorrhoea, pyrexia of unknown origin and Sonne dysentery. The test was made with concentrated suspensions from the Standards Laboratory, Oxford, and account was taken only of "standard" agglutination. The results obtained in the healthy prisoners of war were not significantly different from those in the other cases so that the following combined table can be regarded as giving a fair index of the responses obtained in persons who have not been exposed to the risk of louse-borne or flea-borne typhus.

The high proportion of sera which agglutinate *Proteus OXK* is striking. Stress is laid on the need for investigation of sera from persons from whom strains of Proteus have been isolated. [Presumably

TABLE I.

Result of Weil-Felix test on sera from 100 " normal " subjects.

(Combined results on Groups 1, 2, 3 and 4).

Suspension	Time of reading	No agglutination at 1/8 or over	No. of sera showing "Standard" agglutination at 50°C. in a dilution of:					Percentage showing agglutinin titre not higher than 1/16
			1/8	1/16	1/32	1/64	1/128	
Proteus OX19	4 hrs.	58	19	17	5	1	0	94
	24 hrs.	39	20	22	16	2	1	81
Proteus OX2	4 hrs.	51	29	12	8	0	0	92
	24 hrs.	33	27	21	17	2	0	81
Proteus OXK	4 hrs.	1	0	30	60	7	2	31
	24 hrs.	0	0	15	69	13	3	15

because the organism in these cases would be likely to cause the development of agglutinins and so introduce a possible fallacy in diagnosis.]

In Britain louse-borne and flea-borne typhus are the only fevers of the typhus group that are likely to need consideration so that sera need only be tested against *Proteus OX19,* which is not likely to agglutinate in titres higher than 1–64 after four hours' incubation at 50°C., except in cases of typhus infection. It is stated that " agglutination appears in positive cases at the end of the first week, so repeated examination is advisable if the first sample of blood is taken early during illness or if the titre is not more than 1–64."

John W. D. Megaw.

DAMMIN (Gustave J.) & BILLINGS (F. Tremaine), Jr. **The Weil-Felix Reaction in Patients with Proteus and** *Pseudomonas aeruginosa* **Infections.**—*Jl. Immunology,* 1942. July. Vol. 44. No. 3. pp. 251–258. [11 refs.]

The sera of persons infected with *Proteus mirabilis, P. vulgaris* or *Pseudomonas aeruginosa* usually contain a minor antigen which, on the average, agglutinates *Proteus OXK* in high titre, *P. OX2* in lower titre and *P. OX19* in still lower titre.

" It is not to be implied that the organisms cultured were in each case assumed to have played a major rôle in causing or determining the course of the patient's illness."

The investigation dealt with 14 cases : *Proteus mirabilis* was isolated from 12 patients, usually from the urine, and usually in infections of the urinary tract. From one of these patients *Ps. aeruginosa* was also isolated. *P. vulgaris* was isolated from two patients, one of whom had an abscess and the other an infected wound.

Details are given of the agglutination reactions in all the 14 cases: the following are a few examples:—

Diagnosis	Cultures	Titre of Agglutination			Patient's organism
		Proteus OX19	*P. OX2*	*P. OXK*	
Abscesses	*P. vulgaris*	1–40	1–320	1–640	1–640
Urinary tract infection	*P. mirabilis* (from urine)	0	0	1–80	0
Do.	Do.	1–80	1–80	1–640	1–160
Do.	Do.	1–20	1–20	1–640	1–80
Do.	Do.	1–160	1–5,000	1–2,560	1–320

Only those patients who developed appreciable agglutinative titres to their own organisms showed agglutinins in significant titres for the Weil-Felix antigens. Of ten sera which agglutinated the homologous organism in titres of 1–80 and over, nine agglutinated *Proteus OXK*, three agglutinated *OX2*, and three *OX19*, in similar dilutions. The general response of the positive sera was a moderately low titre for *OX19*, a higher titre for *OX2* and a still higher titre for *OXK*.

The general pattern of the response as compared with that observed in various types of typhus fevers is as follows:—

	Proteus OX19	*Pr. OX2*	*Pr. OXK*
Typhus fever	++++	+	+
Rocky Mountain spotted fever	+++	+++	+
Tsutsugamushi	+	+	++++
Proteus or Pseudomonas infections	+	++	+++

It will be seen that the general pattern of the response to Proteus infections approaches to that of mite-borne typhus.

Sera from three cases of typhus and from one of Rocky Mountain spotted fever were tested for agglutination with the strains isolated from the cases of Proteus infection; there was no response in a titre higher than 1–20.

[This important article must be consulted in original form by workers on the serology of the fevers of the typhus group; the summary gives only the broad outlines of the investigation. Evidently infections with *Proteus mirabilis* are by no means rare and they are likely to give rise to high-titre agglutination of *Proteus OXK*; false positives with *OX2* are less likely to occur, and fortunately *OX19* is seldom agglutinated in significantly high titres.] *John W. D. Megaw.*

MÜHLENS (P.). Die Bedeutung des Fleckfiebers für Afrika. [**Typhus in Africa.**]—*Deut. Trop. Ztschr.* 1941. Apr. 15. Vol. 45. No. 8. pp. 248–256.

This is a useful summary of the geographical distribution of fevers of the typhus group in Africa. Mühlens includes a table of classification of the usual kind. Extensive outbreaks of epidemic louse-borne typhus have been recorded since 1931 in Algeria, Morocco (especially

Marrakesh and Casablanca), Tunis and Egypt. In Cyrenaica there was no record of widespread typhus in this period. It is known in Eritrea and Uganda. In 1934 there was an epidemic in Urundi, and cases have been reported, in smaller numbers, from Kenya and Tanganyika. In West Africa the disease is rare. In South Africa, however, it is a serious problem ; in 1921 there were many thousands of cases in Ciskei and Transkei, and in 1934–35 even more were reported from Basutoland and the Orange Free State. South-West Africa is also infected.

The murine, flea-borne form, is found in North Africa, the Belgian Congo, Freetown, Dakar and in South Africa. Tick-borne typhus is present in the Mediterranean countries, French Equatorial Africa, the Congo, and in South Africa (Swaziland and N. Transvaal especially). The mite-borne form is not known in Africa.

[Since this paper was written louse-borne typhus has spread in severe epidemic form in Morocco, Algeria and Tunis.] *C. W.*

Dormanns (E.) & Emminger (E.). Fleckfieber-Uebertragung von Mensch zu Mensch durch Bluttransfusion im Inkubationsstadium. [**Transmission of Typhus Fever from Man to Man by Blood Transfusion in the Incubation Period.**]—*Muench. Med. Woch.* 1942. June 19. Vol. 89. No. 25. pp. 559–561. With 3 figs.

An attack of typhus fever, fatal on the 5th day, followed the transfusion of 250 cc. blood from a donor who was found later to have been in the incubation stage of an attack of typhus about three days before the onset. The incubation period of the disease in the recipient of the blood was 11 days : the fatal result was largely due to his extremely debilitated condition which resulted from a severe gunshot wound of the leg which had to be amputated eleven days before the transfusion and after five weeks of conservative treatment. The attack differed in no way from what would have been expected if the disease had been transmitted by lice. The Weil-Felix titre was 1–400 in blood taken after death, and typical granulomatous nodules were found in sections of the medulla oblongata. *John W. D. Megaw.*

Lampert. Der zerebrale und der intestinale Fleckfiebertyp. [**Cerebral and Intestinal Types of Typhus Fever.**]—*Deut. Med. Woch.* 1942. May 22. Vol. 68. No. 21. pp. 521–526. With 4 charts.

This article deals with 221 cases of typhus fever treated at a war hospital in Ostrow, Poland, between 9th November, 1941, and 1st March, 1942. The cases fall into two distinct groups, which are discussed separately. In the first group 117 cases occurred up to 4th January, 1942 ; all are classified as being of the " cerebral type." The patients were guards of prisoners-of-war camps in the district and the attacks occurred in spite of delousing and of standing orders that close contact with the prisoners must be avoided. The cases were typical of those seen in virulent epidemics, the total fatality rate being 25·4 per cent. ; if the 35 patients who had been inoculated more than three days before the onset were excluded it was 35·3 per cent. in the remaining 82. It ranged from *nil* in patients under 25 to over 50 per cent. in those over 50.

In two patients there was neither fever nor rash, but the Weil-Felix reaction was positive at a titre of 1–1,600. The rash was pronounced in all but three. In three undoubted cases the Weil-Felix reaction was negative throughout the illness, though in one of these

the "dry-blood agglutination test" was positive. Albuminuria with a few cylindrical casts occurred in many cases. The Weil-Felix reaction was of no significance in prognosis; in one fatal case the titre was only 1–100, in another it was 1–25,000. Except for one alcoholic patient, aged 54, who had only one dose of vaccine two days before the onset there were no deaths among the 35 inoculated persons, although five of them had received only a single dose four or five days before the onset. The age distribution of the inoculated did not differ significantly from that of the others and the exposure to risk of infection seems to have been much the same in both groups. [Unfortunately the kind of vaccine used is not stated; it must have been either Weigl's or Cox's because these are the two types that have been prepared in Germany. The failure of killed vaccine to prevent attacks in the presence of virulent infection and its power to modify the severity of the attacks is in agreement with the experience of workers on Rocky Mountain spotted fever.]

The second group of cases was of a very different kind; 104 persons, none of whom was inoculated, were attacked between 4th January and 1st February, 1942. They are classed as "intestinal" (59 patients) and "mixed" types (45 patients); the latter being said somewhat to resemble the cerebral type. No reason is given for using the name "intestinal" rather than "mild." Most of the patients were soldiers who had just returned from the eastern front and they must have been infected on the journey; some of them were suffering from wounds or frost-bite. There were no deaths and the course of the illness was, on the whole, mild. The temperature ranged about 39°C. and the fever lasted 10 to 14 days. The clinical course often resembled influenza, and the rash was often very sparse and fleeting. The incubation period was usually 14 to 21 days.

The author states that it is an open question whether the mildness of the attacks was due to infection of low virulence or to the treatment which was systematically carried out from the 1st January, 1942, having been gradually evolved during the previous two months.

The thorough nature of the treatment can be judged by the statement that more than 2,500 hot baths were given; more than 40,000 cc. of "kunstblut" (a proprietary preparation of artificial blood consisting of a solution of haemin and gelatine) and more than 200,000 cc. of Ringer's solution were given by "intravenous infusion." More than 80 intravenous transfusions of fresh whole blood from convalescents were carried out. Some confusion is caused by a conflicting statement in another part of the text where it is remarked that subcutaneous injections of Ringer's solution were given because of the danger of too sudden a dilution of the blood by intravenous injections. The theoretical considerations on which the above methods of treatment were based are discussed at length. No special drugs were given.

[The author will not find many supporters for his view that the treatment may have been responsible for the relative mildness of the attacks in the second group of cases. The long incubation period, which could not have been influenced by treatment, suggests that an infection of low virulence was contracted from persons living in endemic areas and suffering from mild attacks. The great severity of the cases in the first group equally suggests the existence of highly virulent epidemics in the prison camps where the patients became infected. Nothing is said about the conditions prevailing in these camps.]

John W. D. Megaw.

WOHLRAB (Rudolf). Chemo- und Serotherapieversuche an Fleck-typhuskranken. [**Investigation of the Chemo- and Sero-Therapy of Typhus Fever.**]—*Klin. Woch.* 1942. May 16. Vol. 21. No. 20. p. 455.

Several sulphonamide drugs had previously been tested, but the results were not convincing. In the present trials a new sulphonamide called "Be 1034," was used. In one group of cases, treated between July and October 1941, the duration of the fever was reduced on the average by 2·6 days as compared with 31 untreated controls, but in another group of 21 cases treated in November and December 1941, when the severity of the disease was greater, the average duration was reduced by only one day and no clinical benefit was observed. Severe vomiting and sometimes also diarrhoea occurred, the case fatality rate was not reduced and complications were not prevented.

Sulphapyridine ("eubasinum") is referred to as having no obvious effect except possibly in connexion with pulmonary complications.

Convalescent serum and animal immune sera were regarded as being of no great value, either as a preventive or curative treatment.

John W. D. Megaw.

REICHS-GESUNDHEITSBLATT. 1942. Mar. 4. Vol. 17. p. 160.—**Instructions for the Campaign against Typhus.** Circular of the Reich Minister of the Interior, dated 13th February, 1942. [Translation 14 typed pp.]

This circular replaces and supersedes a number of separate orders and instructions which have been issued from time to time. It consists of three parts; one contains instructions for delousing and disinfecting, another gives advice to doctors, and the third tells the general public what they ought to know about the disease.

The first part follows the usual lines for the most part, but there are some points of interest. Reference is made to the protective inoculation of medical, sanitary and nursing personnel; it is stated that the vaccine can be obtained from the Robert Koch Institute, Berlin, the Institute for Experimental Therapy, Frankfurt-on-Main, and the Behring Works. Model plans are available for disinfecting and delousing stations. Experiments are now being made with a type of hot-air disinfecting station, consisting of a chamber heated from the sides and arranged so as to allow of complete separation of the clean from the dirty side. The State Building Department is urged to press on with the provision of delousing posts and to help local bodies in their efforts in the same direction, especially in the few districts where stations have not yet been erected.

Although destruction of lice is the essential matter, it should, when practicable, be combined with disinfection because of the possible risk of infection by the excreta of lice in which the virus can survive after disinfestation with cyanide gas. Formaldehyde, in the strength usually employed, does not kill lice.

The life-cycle and bionomics of the body louse are described in some detail; it is held that a knowledge of these is essential for success in delousing. Larval lice can survive for a week without food, adult lice for not more than a week to ten days, but the eggs can remain viable for several weeks at low temperatures.

Lice and nits may be found on floors, walls and furniture, but only in exceptional cases of very heavy infestation; in such cases repeated

disinfestation is needed. For combined delousing and disinfection a mixture of equal parts of water and a 5·0 per cent. cresol-soap solution, having a cresol content of 2·5 per cent., is recommended, or carbolic acid, 1 in 20, can be used. In delousing infested persons the sites most favoured by lice should be vigorously rubbed with paraffin [presumably kerosene] for two minutes, then lathered with soft soap which is washed off after ten minutes.

The second part, intended for doctors, gives a general account of typhus fever on text-book lines. The doctor who visits a known or suspected case is advised to take off his overcoat and put on a gown of smooth material, covering the trunk and arms, and fastening tightly at the wrists. Apart from this the chief point emphasized is that the bed clothes of the patient must be pulled back carefully and not thrown off, lest the lice should be scattered over persons standing by the bed. Protective inoculation is advised; the vaccine is described as being made either from the intestines of infected lice or from cultures grown in the "germinal membrane" of fertile hens' eggs. No mention is made of the originators of these methods; perhaps because Weigl is a Polish worker and Cox an American. The inoculated persons are not fully protected and must continue to take the usual precautions. The reaction is similar to that resulting from enteric inoculation but is less severe. Vaccination must be renewed every year. Emphasis is laid on the employment of attendants who have had attacks of typhus fever, but no mention is made of the importance of selecting youthful persons. In the section on treatment it is stated that there is no specific drug; the importance of watching the circulatory system and of supporting it with camphor and injections of "cardiazol" is emphasized. Convalescent serum is mentioned in terms of very faint praise. Cold-water treatment and ice bags to the head and praecordium are said to be valuable.

When cases occur, preventive delousing of the whole population in the neighbourhood is advised. Suspects need only be deloused, thorough disinfection is not needed.

The third part gives a clear and simple account of the disease for the instruction of the general public.

[The circular as a whole is rather confusing; there are frequent repetitions which suggest that the material has been brought together from a number of different sources.] *John W. D. Megaw.*

Heilig (Robert) & Naidu (V. R.). **Further Experiences on Endemic Typhus in Mysore.**—*Indian Med. Gaz.* 1942. June. Vol. 77. No. 6. pp. 338–342. With 10 figs. (8 on 2 plates).

Fourteen cases of one of the fevers of the typhus group are discussed; these include four cases already described by the authors [see this *Bulletin*, 1942, Vol. 39, p. 375] and diagnosed as "endemic tropical typhus." Ten of the patients lived in Mysore city, the other four came from large villages in the State. Four cases occurred in August, two in September and one in each of the months October to February. There were no cases in the hot months, March to July. The cases were sporadic, only in one instance did two cases occur in the same house. The fever lasted 16 to 21 days, the rash was conspicuous, it was maculo-papular and extended to the face in six cases; the palms and soles were affected. Staining at the sites of the spots persisted for weeks or months and was even more conspicuous in the third and fourth weeks than the rash had been.

In all the previous four cases the Weil-Felix reaction was completely negative, it was also negative in one of the later series, almost negative in another, but in all the remaining six cases in which it was tested the results were positive, as is shown in the table :—

Day of Fever	*Proteus OX19*	*Pr. OXK*	*Pr. OX2*
12	1–40	1–40	1–320
21	1–320	1–320	1–80
10	1–40	1–40	1–640
15	1–160	1–20	1–1,280
22	1–160	1–40	1–160
9	1–80	1–20	1–160
21	1–80	1–40	1–640
35	0	0	0

Blood from one of the patients previously reported had caused a scrotal reaction in guineapigs but in four of the other cases that were tested the results were negative. There was no louse infestation in any case and no history of bites by ticks or mites could be obtained. All the patients lived in extremely close contact with rats and most of them in equally close contact with tick-infested cattle.

Fleas collected from two rats caught near one patient's house were inoculated into guineapigs, which developed fever and reacted to *Pr. OX2*, +1–160. Fleas from rats trapped in two other patients' houses gave negative results. Fifty ticks from cattle belonging to two patients were emulsified and injected into guineapigs with negative results.

The authors confidently exclude louse-borne and mite-borne typhus, they also state that the cases differ in every essential point from the " Bangalore type " which is supposed to be rat-flea typhus. Clinically and serologically the cases closely resembled Boyd's " *X2* " group, which in turn is clinically related to Indian tick typhus, but the author holds that the designation " tick typhus " is proved to be right only for a small minority of Indian typhus cases and that it is doubtful whether the tick is the only vector of " *X2* " typhus.

[In their clinical and serological features these cases conform closely to tick-borne typhus but the epidemiological conditions are more in keeping with the diagnosis of the flea-borne disease.

The association with tick-infested cattle was not mentioned in the previous report ; dogs also deserve consideration. It is just possible that attention may have been focussed too exclusively on transmission by the bites of ticks ; contact with crushed ticks or with their excreta has to be taken into account.] *John W. D. Megaw.*

DE MAGALHÃES (Octavio). **Exanthematous Typhus of Brazil in Minas Geraes. Epidemiology.**—*Jl. Trop. Med. & Hyg.* 1942. Apr. 15 & May 1. Vol. 45. Nos. 8 & 9. pp. 57–61 ; 65–69. With 4 figs. (1 map).

The author has already written a preliminary report on the transmission of the tick-borne typhus fever of Minas Geraes [see this *Bulletin*, 1941, Vol. 38, p. 444]. He now discusses the epidemiology of the disease and gives his general conclusions based on further observations.

Although the chief vector is *Amblyomma cajennense* the bed-bug may perhaps be capable of transmitting infection in natural conditions. Bugs collected from the beds of patients have caused the disease in inoculated guineapigs in some cases. In one case laboratory-bred bugs were fed on a patient and afterwards transmitted infection to *rhesus* monkeys by their bites : later experiments were negative and PARKER failed to transmit the similar disease in São Paulo by bed-bugs. The typhus fever of Minas Geraes persists in certain localities though these may remain quiescent for several years. In one large area where the disease has persisted for a long time there is a zone in which cases have never been seen, probably because periodical burning of the grass destroys the ticks, drives out the reservoir wild animals and makes the place unsuitable for grazing. In this zone there are huts in which a few bugs and ticks exist and dogs are fairly numerous.

Wild animals may bring the Rickettsiae to suburban areas ; alternatively domestic animals may bring infected ticks into the houses. The latter suggestion is favoured by the fact that ill-fed dogs often share their owners' beds. Dogs as well as human beings are regarded as being only transitory and accidental reservoirs of infection. In " dozens of cases " ticks collected from infected persons were found to be infective to animals by immediate or late inoculation. By actual tests the author has excluded mosquitoes, fleas, lice and *Boophilus microplus* [presumably *B. annulatus* var. *microplus* Neumann 1901] from the rôle of possible vectors. There are probably many natural reservoirs of infection, including dogs, domestic and wild cats, foxes, opossums, squirrels, the coati, the capybara and other wild rodents. Wild and house rats have never been found infected.

Quiescence of the disease in known foci is probably explained by the immunity acquired by residents through previous attacks which may have been mild. Of 111 cases observed 23 were in females and 88 in males. Persons of all ages are equally susceptible.

By experimental tests in the laboratory it was found that the ticks must remain fixed on the host for 36 hours or more, otherwise transmission does not occur.

[It does not seem safe to count on so long a period in natural conditions ; no mention is made of the reactivation of the virus by a preliminary blood feed. Reactivation might considerably reduce the time needed for transmission, and the necessity for reactivation may account for the small proportion of cases in which natural infection results from tick bites in heavily infested areas. Natural transmission by bed-bugs can hardly be important in view of the strictly local distribution of the infection as opposed to the universal distribution of the bed-bug.] *John W. D. Megaw.*

BRUMPT (E.) & DESPORTES (C.). Grande longévité du virus de la fièvre pourprée des Montagnes Rocheuses et de celui du typhus de São Paulo chez *Ornithodorus turicata*. [**Prolonged Survival of the Viruses of Rocky Mountain Spotted Fever and the Typhus of São Paulo in** *Ornithodorus turicata*.]—*Ann. Parasit. Humaine et Comparée.* 1941. Vol. 18. Nos. 4–5–6. pp. 145–153. With 4 figs.

Suspensions of the tissues of 4 *O. turicata* infected with the virus of Rocky Mountain spotted fever 4 years, 6 months and 16 days previously were found to be infective to guineapigs, causing typical

febrile and orchitic reaction. In similar conditions the virus of the typhus of São Paulo survived for nearly 5 years; the reaction in guineapigs in this case was mild and produced only a certain degree of immunity.

With both viruses some lots of infected ticks kept for the same length of time were found to be non-infective; it was not possible to estimate the percentage of ticks in which the virus survived for these long periods. The virus of neither of these diseases could be transmitted by the bites of *O. turicata* even when the site was soiled with coxal fluid and excreta.

The authors find it difficult to express an opinion about the epidemiological significance of their experiments. Adult *O. turicata* are too tough to be crushed on the human skin in such a way as to cause infection, but nymphs are more delicate and so may eventually play a part in transmission. The ingestion by guineapigs of *O. turicata* infected with the virus of Rocky Mountain fever did not cause infection, so it appears that this species of tick cannot be regarded as a source of infection in this way. *John W. D. Megaw.*

PERRIN (Theodore L.) & BENGTSON (Ida A.). **The Histopathology of Experimental "Q" Fever in Mice.**—*Public Health Rep.* 1942. May 22. Vol. 57. No. 21. pp. 790–798. With 6 figs. on 2 plates.

A detailed account, illustrated with good photomicrographs, is given of the histological changes found in young Swiss mice at varying periods after intranasal and intraperitoneal inoculation with yolk-sac cultures of the Rickettsiae of Q fever. Nodular and patchy granulomata, composed chiefly of large mononuclear cells, were found in the spleen, liver, kidneys and adrenals of most of the animals. In 52 of 54 mice inoculated by the intranasal route there was an early pneumonic reaction with proliferative changes; large and small mononuclear cells predominated in the exudate. A point of great importance is that pneumonia occurred also in 8 of 20 control mice inoculated intranasally with normal yolk-sac material: the reaction was different, being more exudative than proliferative, and polymorphonuclear cells predominated in the exudate.

The production of pneumonia in the lungs of mice inoculated intranasally with Rickettsiae is, therefore, not in itself evidence of the presence of the virus in the material employed. The report will appeal specially to workers on Rickettsial diseases; it gives a clear statement of the technique employed. *John W. D. Megaw.*

GEAR (James), HARRIS (L. C.) & SANER (R. G.). **Anti-Typhus Vaccination of Guineapigs with Vaccine Prepared from Infected Gerbils.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1942. Aug. 31. Vol. 36. No. 2. pp. 95–98.

In a previous communication GEAR and DAVIS [this *Bulletin*, 1942, Vol. 39, p. 750] showed that the gerbils of South Africa, after preliminary exposure to X-rays, were susceptible to the virus of epidemic typhus, murine typhus, and tick-bite fever. In many cases there were profuse growths of Rickettsiae after intraperitoneal inoculation. Vaccines have now been prepared by the Zinsser-Castaneda method. The exposure to X-rays was for 15 minutes, the total dose being 600 R. One cc. of a suspension—prepared by emulsifying in 15 cc. saline the brain of a guineapig killed on the 3rd day of fever resulting from

inoculation with *R. prowazeki*—was inoculated into the peritoneum of a gerbil: the animal was kept in a room at 70° to 80°F. After an incubation period of 5 to 10 days the animal usually became ill and was moribund within a day or two of the onset.

The peritoneal cavity was first scraped with a blunted and slightly roughened scalpel and then washed out with 0·2 per cent. formol saline. From 20 to 40 cc. of suspension, containing Rickettsiae and cells, were obtained from each animal. By differential centrifugation a deposit of Rickettsiae was obtained; this was made up with 0·1 per cent. formol saline to an opacity corresponding to 1,000 million *Bact. coli* per cc. Details are given of the technique employed.

Six young guineapigs were given three doses of 1·0 cc. each of the suspension at weekly intervals. After a month the animals were found to be immune against large doses of the virus. Five control animals inoculated with the same infecting doses and four inoculated with one-tenth of these doses were all susceptible; so were three of five animals inoculated with 1/100 of these doses.

Several hundred gerbils have been sent to the South African Institute for Medical Research, where the vaccine is now being prepared on a large scale.

Nine laboratory workers have been vaccinated with four doses, of 1·0 cc. each, at weekly intervals. Slight local reactions, less than those with T.A.B. vaccine, followed in eight persons; in the ninth there was a history of allergy and a severe local reaction resulted from the second dose; the later doses were given intradermally and the reactions were much less. There were no general reactions except that one of the inoculated had slight headache on the day following the second injection. *John W. D. Megaw.*

BARTONELLOSIS.

HERTIG (Marshall). **Phlebotomus and Carrion's Disease. I. Introduction. II. Transmission Experiments with Wild Sandflies. III. Field Studies on Phlebotomus. IV. Massive Infections of the Sandfly Proboscis with Unidentified Microorganisms.**—*Supplement to Amer. Jl. Trop. Med.* 1942. July. Vol. 22. No. 5. 81 pp. With 14 figs. on 3 plates.

This monograph, which is divided into four sections, is a valuable summary of research work on the rôle of Phlebotomus in Carrion's disease, accomplished during the past five years in the Instituto Nacional de Higiene y Salud Publica, of Lima, Peru, in collaboration with the Department of Comparative Pathology and Tropical Medicine of the Harvard University Medical School. Part of the data presented has already been published [this *Bulletin*, 1938, Vol. 35, pp. 375 376; 1940, Vol. 37, p. 853; Proc. VI Pacific Sci. Congress, 1939, Vol. 5, pp. 775–779; Proc. III International Congress for Microbiology, New York, 1939, pp. 401–402]. In the introductory section is given an excellent historical review of entomological investigations relating to Carrión's disease and a summary of the views which have been put forward as to the method of transmission of the infection.

In Section II, the author records detailed results of transmission experiments in the laboratory with wild-caught Phlebotomus from

endemic zones. It is pointed out that NOGUCHI and his collaborators [this *Bulletin*, 1929, Vol. 26, p. 849] and BATTISTTINI [this *Bulletin*, 1932, Vol. 29, p. 30] showed that *Bartonella bacilliformis* could be recovered in *rhesus* monkeys from Phlebotomus sandflies taken in an endemic area. In addition, the latter investigator succeeded in infecting a monkey by releasing sandflies in its screened cage, though the method of infection, whether by bite or otherwise, was not determined. In the first series of experiments described in this report, three monkeys bitten by 159 out of 627 sandflies, and two monkeys which were injected with suspensions of 340 or more sandflies, mostly from Verrugas Canyon, did not develop the disease. In the second series of tests eight *rhesus* monkeys were exposed to the bites of wild-caught sandflies. Five of these, bitten by 89 out of 447 sandflies (*P. verrucarum*) became infected with *Bartonella bacilliformis*. No skin lesions were produced. Infection was demonstrated by blood cultures, subsequent tests of such cultures in monkeys, and in two instances by immunity tests. The sandflies were placed in small feeding-cages and applied to the skin of the monkeys in such a way as to permit voluntary and natural feeding. While other mechanisms of transmission are theoretically possible, the author feels reasonably certain that transmission occurred by the bites of the sandflies. Several species of South American monkeys were infected by inoculation of cultures. Nodules were produced from which *B. bacilliformis* was recovered in culture, though blood cultures were negative.

The results of extensive field studies on the three species of Phlebotomus conducted throughout more than four years at different places and altitudes in the Rimac and Santa Eulalia Valleys, are presented in Section III. The author lays down more accurate criteria for the identification of females of *P. verrucarum* and *P. noguchii*. This matter is of importance chiefly because NOGUCHI and his colleagues produced infections in monkeys with sandflies thought to be *P. noguchii* [this *Bulletin*, 1929, Vol. 26, p. 849]. It now appears that this identification was an error and that *P. verrucarum* is probably the most important transmitter to man.

In the last section of the monograph Hertig describes a curious infection of the tip of the proboscis found in both males and females of the three species of Phlebotomus captured in the Peruvian verruga zone. Over 300 such infections have been observed. *B. bacilliformis* has been recovered in culture twice and an unnamed micro-organism of similar morphology about thirty times. The proportion of sandflies infected is frequently as high as 40 to 50 per cent. In many cases the pharynx is also infected. The source of the proboscis infection is unknown. Artificial feeding of *P. verrucarum* with cultures of *B. bacilliformis* has failed to produce comparable infections of the proboscis. Since the condition has been found in males, which do not suck blood, and in females which have never had a blood meal, the possible relation of this infection to Carrión's disease remains obscure.

[The importance of Carrión's disease as a public health problem has been greatly emphasized by the recent discovery of the infection in Ecuador and the occurrence during the past few years of an extensive outbreak in the Department of Nariño in Colombia. There is, therefore, an urgent need for additional research of the careful, patient type, such as that upon which this monograph is based, to furnish an adequate picture of the epidemiology of the disease.]

Hugh H. Smith.

Ristorcelli (A.) & Dao Van Ty. Phlébotomes d'une région de Colombie où la verruga du Pérou est devenue endémique depuis trois ou quatre ans. [**Phlebotomus in an Endemic Area of Verruga Peruana in Colombia.**]—*Ann. Parasit. Humaine et Comparée.* 1941. Vol. 18. Nos. 1–2–3 & 4–5–6. pp. 72–74 ; 251–269. With 9 figs. & 2 plates. [12 refs.]

YELLOW FEVER.

Nauck (E. G.). Die Bedeutung des Gelbfiebers für Afrika. [**The Importance of Yellow Fever in Africa.**]—*Deut. Trop. Ztschr.* 1941. May 1. Vol. 45. No. 9. pp. 272–277.

Teesdale (C.). **Pineapple and Banana Plants as Sources of Aedes Mosquitoes.**—*East African Med. Jl.* 1941. Dec. Vol. 18. No. 9. pp. 260–267.

Mosquitoes were found breeding around Mombasa in collections of water in pineapple and banana plants. The commonest species in them was *Aëdes simpsoni*, but *Aëdes aegypti*, *Aëdes metallicus* and *Aëdes pembaensis* were also found. Larval development was sometimes extremely slow [probably on account of the lack of food in the water]. The banana trees hold water throughout the year ; an adult *Aëdes simpsoni* emerged from a banana plant at the height of the dry season. This may help in the persistence of adults until the next rains.

V. B. Wigglesworth.

DENGUE.

Singh (Bhupal). **Seven Days Fever (?).**—*Jl. Indian Med. Assoc.* 1942. July. Vol. 11. No. 10. pp. 303–307. With 13 charts.

In Meerut [near Delhi] a number of cases of short fever were seen during September, October and November 1941. The onset was sudden, with high temperature, severe headache and pains all over the body, but specially in the calves and back. The pulse was unusually slow in relation to the temperature and in the cases examined there was leucopenia with lymphocytosis. Some of the temperature charts showed a peculiar saddle-back type, others a continued type.

The author briefly discusses the differential diagnosis between the " seven day fever " described in India 34 years ago and the seven day fever of Japan, but expresses no definite opinion about the nature of the outbreak.

Thirteen charts are shown. [With one possible exception in which the fever lasted eight days the curves are of types frequently seen in dengue, and the description of the disease is in all respects in keeping with the diagnosis of dengue.] *John W. D. Megaw.*

Chaudhury (L. M.) & Ghosh (S. M.). **A Fever of Seven Days' Duration at Patna.**—*Jl. Indian Med. Assoc.* 1942. June. Vol. 11. No. 9. pp. 269–273. With 3 figs.

This outbreak, in which 50 cases of fever lasting six or seven days were studied at the Medical College Hospital, Patna, in September

and October 1941, is clearly of the same type as the one just reviewed. The authors regard the disease as a definite clinical entity of obscure aetiology and differing from dengue in the absence of break-bone pains, rash, and division into three stages of invasion, remission and terminal fever. It differs from Japanese seven day fever in its epidemiology, the absence of glandular enlargement and leucocytosis, and in the failure to isolate leptospirae from four animals inoculated with the blood of patients.

[The claim that the disease "differs from other fevers of short duration" must be scrutinized. Three fever curves are shown: two of these would serve as good illustrations of characteristic dengue curves, and the third is quite in keeping with the diagnosis of dengue. Repeated leucocyte counts were made only in one case, in which the total count fell from 8,000 on the third day to 4,500 on the fifth day. The days of the disease on which the other counts were made are not specified, so that it is impossible to form an opinion about their significance: the lowest count was 3,200, in 13 cases it ranged from 4,200 to 5,000, in 11 between 5,100 and 6,000, in 11 between 6,100 and 7,000, and in 6 it was over 7,000. It exceeded 9,500 in two cases, in one of which it was 13,400 and in another 16,200, so that probably there was some complicating factor to account for these anomalous findings.

Apart from these two cases the other counts are such as could be expected in a series of examinations made at various stages of attacks of dengue. The absence of a rash is the only feature in which the outbreak can be held to differ from the generally recognized picture of dengue; but in many outbreaks of undoubted dengue among dark-skinned persons living in places where the disease is endemic the rash has been conspicuous by its absence; even in fair-skinned persons it may occur in only 5 to 10 per cent. of the cases.

In all its other features the disease corresponds to dengue. Fifty-four per cent. of the patients were medical students or members of the staff of the hospital, blood cultures were invariably sterile, there was no evidence of infection by personal contact, and there were no deaths. Body-ache is mentioned as a special feature of 19 of the cases. The authors make no reference to the "seven-day fever of India" which is closely similar and which is now regarded by its sponsor to be a form of dengue. This paper and the one by SINGH are reminiscent of the controversy of more than 30 years ago in Calcutta; they show that there is still a reluctance to diagnose dengue when an outbreak does not conform in every detail to the descriptions of the classical epidemics.]

John W. D. Megaw.

PLAGUE.

MEYER (K. F.). **The Ecology of Plague.**—*Medicine.* 1942. May. Vol. 21. No. 2. pp. 143–174. [50 refs.]

Wild rodent reservoirs of plague are now in existence in 12 Western States of North America and at least one province in Canada. One of the most important discoveries with regard to plague has been the existence of rodent infection not only in the rat but also in ground squirrels. Both of these rodents may occupy the same burrows and their "ectoparasites

are mutually exchangeable." Although this has been known for some time it is not always easy in practice to obtain evidence of the infection of ground squirrels. In one county, for example, as many as 8,000 squirrels were examined before positive infection came to light. Between 1908 and 1927 the United States Public Health Service reported 2,069 proven plague infections among some 558,706 squirrels. "Thus an animal survey catch of 30,000 rodents should produce, if the disease is spread uniformly over the endemic area and occurs yearly with the same intensity, an average of 15 plague infected animals." In typical plague years this figure has usually been greatly exceeded.

In California tularaemia may cause extensive epizootics among rabbits and hares, but it is important to know that under these circumstances "only an occasional squirrel is similarly diseased." In fact "in recent years every true epizootic among . . . . squirrels was invariably proven to be caused by *P. pestis*." At first search was made for rodent plague in animals which had been shot and dissected, until it was found that pools of fleas collected even from healthy animals might prove the existence of endemic plague foci when tested on guineapigs.

Although the ground squirrel is perhaps the most important host of sylvatic plague in California it is not by any means the only rodent thus incriminated. Plague is enzootic over at least 12 Western States and "not less than 31 species of wild rodents form a reservoir of plague independent of the domestic rat." Table 3 gives a full list of these rodents. In the squirrel the plague lesions resemble more those found in the infected guineapig than in the rat. "A bubo with a purulent focus in the inguinal region is noted in about four-fifths of all cases . . . . In the acute stages the spleen is invariably very much enlarged . . . while in the subacute form caseation or purulent lesions in . . . liver, spleen and lungs are noted. Towards the end of an epizootic period or in an enzootic area rodents with one lymph node containing small abscesses and very few bacilli are encountered. This form of infection is known as the residual type . . . ."

Much important information is now being collected by the staff of the Hooper Foundation on plague rodents and fleas. Some of this is already recorded in this work. The susceptibility to plague of a few species and of individuals of the rodent population has been tested. In all probability in the various species the range of susceptibility, or acceptance of plague experimentally, is not very great. On the other hand considerable difference may be found among the individuals of a given rodent population and the percentages of susceptible and resistant individuals, especially in proven and non-proven plague areas, may vary significantly. Furthermore, the tests made "confirm the epidemiologic fact that the susceptibility of the young immature rodents and liability of the mature males to *P. pestis* infection is much higher than that of mature females."

Flea population studies have been vigorously pursued: "over 50 different species have been found on the Western wild rodents (Eskey and Haas)." A subject of considerable importance for sylvatic plague in the bionomics of ectoparasites is that of "the influence of climatic conditions on the life and longevity of the insects. The 'microclimate,' the temperature and humidity of the burrow, exert their influences on the life cycle of the flea and thus on the death rate of the insect . . . ." Certain terms relating to infection by fleas are defined. Among wild rodent fleas it is found, just as in the case

of the rat flea *Xenopsylla cheopis*, that only a few of the "infected" fleas become "infective." Thus we have the term "vector potential" defined as "the percentage of infected individual fleas to become infective" and again "transmission potential," as the mean number of transmissions effected by a group of infective individuals. "From the preliminary studies on three species of fleas it is recognized that neither a high 'infection potential,' namely the acceptance and implantation of the *P. pestis* in the alimentary canal of the insect, nor the vector potential alone without a consideration of the transmission potential is indicative of high vector efficiency."

The last subject treated in this article is that of rodent plague epizootics. In some counties these are very localized and foci may act as individual units without sign of spread even to other localities not more than 15 to 20 miles distant. "Focal occurrence and discontinuous distribution is apparently one of the characteristics of sylvatic plague." One of the most interesting research studies requiring intensive investigation is that of the mechanism of the *persistence or perpetuation of rodent plague infection* and the seasonal "carry over" of this infection.

This summary of an important article can only be described as sketchy: the author describes even the article itself as only a "sketchy survey of the ecology of sylvatic plague." *W. F. Harvey.*

DOWDESWELL (R. M.). **Estimation of the Value of any New Treatment in Disease with Special Reference to Plague.**—*East African Med. Jl.* 1941. Dec. Vol. 18. No. 9. pp. 258–260.

The author briefly states the arguments in favour of controlled experiment in the application of any new form of treatment or prophylaxis to man, provided that there is a *prima facie* case in favour of the new method and that it is not certain that it is overwhelmingly better than the old. He quotes figures of plague case mortality rates: In Kenya, from 1930 to 1937 this was 59·5 per cent. of 479 patients. For the value of preventive inoculation the more important statistics up to 1936 showed fatality rates of 79 per cent. in the uninoculated and 48 per cent. in the inoculated. In India, WAGLE *et al.* [this *Bulletin*, 1941, Vol. 38, p. 628] recorded a case mortality rate of 21 per cent. in patients with bubonic plague treated with sulphapyridine and sulphathiazole, and of 43 per cent. (as against 95 per cent. in controls) of those with septicaemia so treated. *C. W.*

CHOLERA.

TAYLOR (J.). **Cholera Research in India 1934–1940 under the Indian Research Fund Association. A Review.**—38 pp. 1941. Cawnpore: The Job Press.

There is no doubt about the usefulness of this brochure which, although entitled Cholera Research in India, deals in reality with the whole cholera problem. It has the added value that the author gives an opinion in no uncertain terms on what is cholera, whether it be sporadic or epidemic. The discussion is bound up with those most important questions: What is the true cholera vibrio, what are haemolytic vibrios, what is the El Tor vibrio, and what is the new recruit

to the cholerigenic class, the Celebes vibrio? These are questions of international interest. Emphasis has been laid on the serological identification of cholera. Evidently the project for establishing a "Standard Agglutinating Serum" could not be realized without more evidence, procurable only by testing in the field. An obvious field for the purpose was that in which cholera was endemic and assumed epidemic form. Truly endemic cholera centres are to be found in Lower Bengal, India and the Yangtse Valley, China, but other areas are probably endemic. It is interesting to have the basis laid down for recognition of an endemic area as (1) one in which the total number of months with absence of cholera deaths shall not exceed 30 in 32 years, and (2) in which no break of 5 months or more in cholera incidence shall take place.

In the investigation of the serology of vibrios standard O antigen was used. This was prepared in dry form and testing sera were prepared for local use from this dried antigen. The two main subtypes of *V. cholerae* were used, the "original" and "variant" types or, as they are more commonly called, the Inaba and Ogawa strains. These contain a common O antigen, and in addition each contains its own specific O antigen. Strains containing all three O antigens, the "intermediate" type, were not used. Serum for testing was also prepared against an unheated suspension of a selected *V. cholerae* strain (H+O serum). A very important part of the Indian enquiry was the determination "whether vibrios of serological type other than O group I could be assigned a cholerigenic role" and perhaps the most important verdict reached on this question is given in the pronouncement that: "The incidence of vibrios inagglutinable with O group I serum in cases of clinical cholera in Calcutta would appear to be no greater than in the general population of the area." That result, very nearly if not wholly, indicates that vibrios isolated from true cholera cases, but which do not conform to the serological O group I, are mere accompaniments of the true cholera vibrio and are intestinal inhabitants as much of the non-cholera as of the cholera population.

In India the El Tor type of vibrio has been regarded as non-existent and the only exception to this statement has been found in certain water vibrios in Bengal. Only slight variation seems to have been adopted in the technique of GREIG for determining haemolytic power with goat erythrocytes. "The essential difference between the Greig-positive and Greig-negative strains found was that fresh cultures of the former gave rapid lysis under all conditions of test while the latter only gave slow lysis in the presence of adequate oxygen and warmth." [See READ *et al.* below.]

Much attention has been focussed on the biochemical reactions which form the basis of HEIBERG'S classification of vibrios. The three-sugar differential characterization of O group I vibrios as mannose +, arabinose −, saccharose + is not really differential, although if combined with positive cholera-red and negative Voges-Proskauer reactions it may justify a presumptive diagnosis of *V. cholerae*.

Attempts at forecasting cholera do not appear to have been very successful, while it is of great importance to realize that the "carrier" such as is known in typhoid fever does not exist for cholera. The position may be summed up as: (*a*) "The cholera convalescent and the contact carrier in most cases are free from the vibrio after five days from onset of the attack or contact with a connected case . . . (*b*) Persistence of *V. cholerae* in water is apparently longer, the

maximum found . . . being 16 days. (*c*) The cholera vibrio has not been isolated except in immediate relationship to the cholera case." Thus the cholera case itself appears to be the major factor in disseminating infection and maintaining endemicity, while close contact carriers and water sources infected from the case act only as intermediaries for short periods and at short range.

This " review " should be read carefully for further details.

W. F. Harvey.

READ (W. D. B.), PANDIT (S. R.) & DAS (P. C.). **Action of *V. cholerae* and El Tor Type Strains on Goat's Red Corpuscles.**—*Indian Jl. Med. Res.* 1942. Apr. Vol. 30. No. 2. pp. 183–212. [10 refs.]

The test of haemolytic power in vibrios, established by GREIG in 1914 [this *Bulletin*, 1915, Vol. 6, p. 36], has remained valid and is very largely used for the separation of the true classical cholera vibrio. It depends on the power of plain broth cultures of vibrios to haemolyse goat erythrocytes. The authors have modified the test by substituting tryptic digest broth for plain broth and making it isotonic by using 0·85 per cent. instead of 0·9 per cent. salt in its composition. They divide vibrio strains into " Greig-positive " and " Greig-negative " according as they haemolyse within 24 hours or not. These groups correspond to " early haemolytic " and " late haemolytic." Haemolysis in the latter group is usually partial and never takes place within 24 hours.

A large number of strains was tested and comprised Indian and Celebes vibrios with a few classical El Tor strains. They were tested under aerobic, partial aerobic and anaerobic conditions ; at incubation temperatures of 37°C. and 12°C. ; with 2·5 per cent. and 0·5 per cent. erythrocyte suspensions ; with organisms grown for 1 to 15 days, and in other ways. Under all these conditions the Greig test for the haemolytic vibrio was supported. An important difference between the " early haemolytic " and the " late haemolytic " groups was that the haemolysin of the former was specifically neutralized by an antiserum prepared from Greig-positive organisms, whereas no similar effect was demonstrated in the late haemolytic group. " The ' early ' and ' late ' haemolysins are very likely identical with the haemolysin and the haemodigestive ferments respectively, described by van Loghem in respect of solid blood media." [See this *Bulletin*, 1914, Vol. 3, p. 480.] *W. F. Harvey.*

CARRUTHERS (L. B.). **Sulphanilylguanidine in the Treatment of Cholera.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1942. Aug. 31. Vol. 36. No. 2. pp. 89–93.

In an epidemic of 138 cases of cholera, 50 patients received sulphanilylguanidine in addition to intravenous hypertonic (twice normal) saline and relatively large amounts of sodium bicarbonate by mouth. The remaining cases provided the controls, and in these sulphanilylguanidine was not given. The initial dose of the drug was 0·1 gm. per kgm. of estimated body weight and the maintenance dose 0·05 per kgm. every four hours until the stools became normal. " The stools were considered to be normal when they had become faecal in character, semi-solid or solid . . . and were not more than two each 24 hours . . . No toxic symptoms . . . were seen . . . It may be

concluded that sulphanilylguanidine has no demonstrated value in the treatment of cholera." There were 7 deaths in the sulphanilylguanidine group (50 cases) and 15 in the control group (88 cases); the difference is not of value. *W. F. Harvey.*

PANJA (G.), MALIK (K. S.), PAUL (B. M.) & GHOSH (S. K.). **Treatment of Cholera with Pyrogen-Free Saline.**—*Indian Med. Gaz.* 1942. May. Vol. 77. No. 5. pp. 282–283.

It had been found that cholera cases treated with hospital hypertonic saline were getting severe rigors and pyrexia. The experiments of the authors were directed to obtaining pyrogen-free distilled water with which to make up the hypertonic salt solution. This was obtained by redistilling distilled water in an all-glass still in the presence of a few drops of strong sulphuric acid to make it faintly acid to litmus paper and a few crystals of potassium permanganate to give it a faint pink colour during the whole process of distillation. Out of 30 cholera patients treated with this hypertonic saline, three developed rigor only and one both rigor and high temperature, as compared with one and three respectively out of 18 treated with freshly distilled water saline, and 5 and 12 respectively out of 18 treated with ordinarily prepared hospital saline. "Pyrogen-free saline can safely be administered in cases with a high rectal temperature." *W. F. Harvey.*

AMOEBIASIS.

ANDREWS (Justin). **The Transmission of *Endameba histolytica* and Amebic Disease.**—*Southern Med. Jl.* 1942. July. Vol. 35. No. 7. pp. 693–699. [66 refs.]

This paper is a review of published work and does not contain any original observations. The knowledge of this matter is meagre and unsatisfactory. Reservoirs of amoebic infection are widely distributed, and probably constitute in America 5–10 per cent. of the population, being most numerous in the south, less so in the north-eastern region.

Transmission occurs most frequently by polluted water, by means of insanitary methods of excreta disposal, personal contact with carriers, insect transmission and contaminated food. Epidemic transmission may be due to water, or to flies.

Factors concerned in production or suppression of symptoms appear to be temperature, the strain of *E. histolytica*, rapid passage, dietary effects and acquired immunity.

[These conclusions are based upon an extensive review of the literature.] *P. Manson-Bahr.*

SCHULZE (William) & RUFFIN (Julian N.). **The Clinical Aspects of Amebic Dysentery as seen in North Carolina.**—*Southern Med. Jl.* 1942. July. Vol. 35. No. 7. pp. 699–705.

The purpose of this paper is to draw attention to the prevalence of amoebic dysentery in North Carolina, the ease with which diagnosis can be made by proctoscopic examination in the majority of cases, and the prompt and dramatic response to treatment.

Amoebic dysentery may occur at any age and is more prevalent in males. Symptoms accompanying diarrhoea are usually mild, occasionally severe; the course is chronic with frequent relapses. Physical examination is of little value in establishing diagnosis; proctoscopy and stool examination are all-important.

The ulcers seen at proctoscopy are so characteristic as to be diagnostic and almost always mucus removed from the ulcer will reveal the presence of trophozoite forms of *E. histolytica*. Other laboratory investigations appear to be of little value and the blood picture is not characteristic.

Radioscopy of the colon, though sometimes suggestive, is rarely, if ever, diagnostic. Complications in this series of 95 cases were noted in 13 per cent.; they were either liver or lung abscesses; the latter may develop in the absence of diarrhoea or even of any suggestive intestinal history.

In the experience of the authors carbarsone by the mouth and chiniofon (quinoxyl) by retention enema proved most effective, but relapses were common (36 per cent.) regardless of the type of therapy employed. The mortality of the whole series was 7 per cent.

The question of diet as a predisposing factor in amoebiasis is discussed. It is emphasized that only two patients could be found who had been subsisting on a well-balanced diet. The remainder were of the tenant farmer class and were known to have subsisted on a deficient diet. *P. Manson-Bahr.*

Ochsner (Alton) & DeBakey (Michael). **Surgical Amoebiasis.**—*New International Clinics*. 1942. Vol. 1. (n.s. 5). pp. 68–99. With 6 figs. [125 refs.]

The object of this symposium is to draw attention once more to the lesions which *Entamoeba histolytica* may produce—manifestations easily confused with other conditions. The fallacious impression still appears to exist that amoebiasis is principally a tropical or sub-tropical disease, whilst the contrary is the case. The incidence of infection in the U.S.A. is well over 11 per cent.

On the basis of Craig's estimate, between six and twelve million people in U.S.A. are infected with *E. histolytica*, whilst the potential number of hepatic complications is approximately between three to six hundred thousand; amoebic hepatitis represents only one-half of the surgical complications.

Amoebic lesions of surgical import can be classified as follows:—

I. Intestinal lesions:
   (a) Appendicitis.
   (b) Perforation with peritonitis.
   (c) Massive haemorrhage.
   (d) "Amoeboma" (amoebic granuloma).
   (e) Cicatricial stenosis.
   (f) Pseudopolyposis.

II. Extra-intestinal lesions:
   (a) Hepatic abscess.
   (b) Pleuropulmonary affections.
   (c) Cerebral abscess.
   (d) Cutaneous ulceration and abscess.
   (e) Splenic abscess.
   (f) Genito-urinary affections.

As regards appendicitis, it is stated that in the Tulane Surgical Service, in which stool examinations are performed on every patient with a tentative diagnosis of appendicitis, some 10 per cent. have amoebiasis and are relieved by antiamoebic therapy. The clinical manifestations are frequently indistinguishable from other forms of appendicitis. Surgical intervention is not only undesirable but may even be dangerous, because such infection is rarely limited to the appendix, but usually also involves the caecum. This was clearly demonstrated by the appallingly high mortality rate of appendicectomy following the Chicago epidemic.

The most serious complication of intestinal amoebiasis is perforation of the bowel. It has been estimated to occur in 1·5 per cent. of clinical cases. It is more likely in the severe, rapidly progressive and fulminating types of amoebiasis.

In such cases, surgical intervention can accomplish little, but in localized peritoneal infections with walled-off peritoneal abscesses, especially in the vicinity of the caecum and sigmoid, the prognosis is better. An illustrative case is cited where fatal peritonitis supervened after a gynaecological operation for removal of fibroids. Massive haemorrhage occurs rarely, most usually associated with the rapidly progressive and fulminating types of infection.

An "amoeboma" (amoebic granuloma) consists of a firm, nodular, granulomatous inflammatory mass with multiple small abscesses, occurring most frequently in the caecum or in the sigmoid. The clinical significance lies in the fact that it may be easily confused with tuberculous, actinomycotic or malignant growths.

The use of amoebicides is important even though surgical intervention may subsequently be necessary. A case is quoted of amoeboma of rectum admitted with tentative diagnosis of carcinoma. Diodoquin was employed as an amoebicide, but proved ineffective and the patient subsequently succumbed to intestinal obstruction.

Cicatricial stenosis of the large intestine may occur, particularly in patients with recurrent infections over long periods of time resulting in destruction of the bowel wall and consequent fibrosis. Such lesions are commonly limited to the caecum and sigmoid, though occasionally they may involve the rectum. An illustrative case is quoted in which a rectal stricture was found 5 cm. above the anus in a woman who had suffered continuously from amoebic dysentery for five years. The obstruction was relieved by injections of emetine hydrochloride.

The survey of extra-intestinal lesions is very thorough, and especially in the case of hepatic abscess follows the generally accepted lines.

P. Manson-Bahr.

D'Antoni (Joseph S.). **Amebiasis. Recent Concepts of its Prevalence, Symptomatology, Diagnosis and Treatment.** —*New International Clinics.* 1942. Vol. 1. (n.s. 5). pp. 100–108. [16 refs.]

This paper contains no original work but summarizes the data upon which the importance of amoebiasis as a disease factor in the United States is based.

From a summary of published series of post mortem reports it is evident that amoebic lesions are predominantly present in the caecum and appendix, rectum and sigmoid.

A logical classification would seem to be :—

1. *Asymptomatic amoebiasis.*
2. *Symptomatic amoebiasis.*
   (a) Asyndromic (formes frustes). Mild toxaemia and vague gastro-intestinal irritation.
   (b) Syndromic. Symptoms simulating chronic appendicitis, peptic ulcer, chronic cholecystitis with lesions confined to caecum.
   (c) Dysentery (acute or chronic).
   (d) Hepatitis and liver abscess.
   (e) Involvement of other organs.

P. Manson-Bahr.

BERKMAN (John M.) & MAGATH (Thomas B.). **Amebiasis. Report of an Unusual Case.**—*U. S. Nav. Med. Bull.* 1942. Jan. Vol. 40. No. 1. pp. 159–165. With 3 figs. (2 on 1 plate).

In this case the patient, after a long period of vague ill-health, with pain across the shoulders and loss of weight, developed moderate pyrexia with continuous leucocytosis (about 15,000), enlargement of the liver, elevation and fixation of the diaphragm and extensive but indeterminate X-ray changes at the bases of both lungs. Exploratory laparotomy was performed and subacute appendicitis, with thickening of the adjacent caecum and mesentery of the appendix, was found. On this amoebiasis was suspected and after the operation the patient was treated with emetine ; the temperature fell rapidly to normal and symptoms cleared relatively quickly. *Entamoeba histolytica* was found in sections from the excised appendix.

It is noted that two stool examinations had previously been negative and that exploration of the liver at operation failed to reveal any sign of abscess. Amoebiasis should be suspected in such cases, and if excised material is examined, a considerable number of sections should be cut, since in this case some were negative while others contained abundance of amoebae. If the material is placed in formalin it can be re-fixed in corrosive sublimate with acetic acid and satisfactory staining can be carried out with iron haematoxylin. The pathology of the enlarged liver is not clear, but under treatment with emetine and treparsol the enlargement subsided.

It is thought probable that the patient acquired her infection during the Chicago outbreak of 1933, but she had no definite symptoms of amoebic dysentery at the time. *C. W.*

BAUMAN (Louis). **An Unusual Case of Amebic Hepatitis.**—*Amer. Jl. Med. Sci.* 1942. July. Vol. 204. No. 1. pp. 105–107. With 1 chart.

"1. Acute abdominal symptoms and signs associated with fever and leukocytosis were erroneously diagnosed as appendicitis.

"2. Removal of a normal appendix was followed by jaundice, abdominal distention, tenderness over the liver and fluid in the right chest. A low serum phosphatase was associated with high serum bilirubin.

"3. Amebae were found in the stool.

"4. Three courses of emetine administration were necessary to effect a cure of the amebic hepatitis."

WESTPHAL (Albert). Zur Frage der Mischinfektion von Amöbenruhr und Typhus. [**The Question of a Mixed Infection of Amoebic Dysentery and Typhoid.**]—*Deut. Trop. Ztschr.* 1942. May 15. Vol. 46. No. 10. pp. 258–264.

The author originally contracted *E. histolytica* infection in May 1938 during experimental work on amoebiasis. The original strain was of Brazilian origin from a child who was a symptomless carrier of the organism. One year later (1939) in San Domingo, whither he had gone to observe the effects of climatic changes on his parasite, he contracted typhoid fever, and so was able to make observations in his own person on this double infection. In Westphal amoebiasis declared itself as a symptomless lumen-infection with *minuta* forms and cysts, and his faeces had served as material for experimental work by B. J. BÖE (1939).

Already in 1937 he had been interested in the effects of a superadded bacterial action in provoking "amoebic dysentery" in the carrier state and during this experiment had succeeded in producing acute enteritis and appearance of amoebic dysentery; later he undertook, with MARSCHALL, experimental work on cats in the same direction; from this it appeared that in these animals the bacterial infection, which was in fact a septicaemia, facilitated penetration of the bowel wall by the amoebae after an incubation period of twenty-three days. It was also possible to contrast his self-inflicted experiment with that of KUNERT (1937) under comparable circumstances, but in this latter case typhoid infection caused acute amoebic dysentery with blood and numerous *E. histolytica*.

There is a further interest inasmuch as Westphal was treated during the attack of typhoid with pyramidon, but in Kunert the amoebic infection was suppressed with yatren without in any way influencing the fever.

It was fully expected that in a typhoid infection which ran its full course, conditions would be produced which would stimulate the *minuta* lumen infection to invade the bowel wall, or would, at least, render local conditions in the mucosa favourable for their entry. In Kunert's case acute exacerbation of amoebic dysentery was noted one week after the diagnosis of typhoid had been established, though no exact observations were made to find out whether the *minuta* became converted into the tissue-invading forms of *E. histolytica*; but in Westphal's case during the whole course of the illness no alteration in numbers or character of *minuta* amoebae or cysts was observed; nor did the stools ever contain blood or mucus.

From the therapeutic aspect, therefore, the typhoid exerted no influence upon the amoebic infection, nor did the amoebiasis influence the typhoid, so that the end of the illness found him in the same state as before with a lumen infection with *minuta* forms and cysts. In Kunert's case apparent cure of amoebiasis with yatren did not influence the course of the typhoid. An explanation of this anomaly can be made on the assumption of varying virulence in different strains of *E. histolytica*. In the parallel studies in cats it was found that the banishing of the typhoid-like bacterial lesions in the bowel was followed by simultaneous healing of the amoebic ulcers. The author believes the

"incubation period" to be important in so far as it depends upon local necrotic damage to the mucosa by subacute bacterial infection, which forms a *locus* for the penetration of the amoebae into the bowel wall; but possibly in acute infections, such as typhoid, other influences are brought to bear which counteract this. *P. Manson-Bahr.*

MOHR (Werner). Bedeutung und Möglichkeiten der Röntgenuntersuchung bei Amöbenruhr und ihren Folgezuständen. [**Radiological Investigation of Amoebic Dysentery and its Sequelae.**]—*Deut. Trop. Ztschr.* 1941. July 15. Vol. 45. No. 14. pp. 417–427. With 12 figs.

Introduction of the mucosal relief technique in radiology has opened up new possibilities in diagnosis of intestinal amoebiasis. As a basis of his studies Mohr subscribes to the thesis of WESTPHAL that bacterial symbiosis is necessary for the production of necrotic foci in the mucosa so as to enable entamoebae from the lumen to penetrate the muscularis mucosae.

Preparation of the patient is important. A cleansing enema is given 2–3 hours before and the barium enema is preceded by proctoscopy, which not only presents a picture of the nature and extent of the ulceration, but also ensures that faecal evacuation has been completed. Care must be exercised against over-inflation of the bowel which may spoil contrast relief.

In the early phases of amoebic dysentery changes in this mucosal pattern are slight and consist of flattening of the mucosal folds and a blurring of the outline, but with more extensive lesions "cushion-shaped" filling defects become visible with irregularities in the pattern of the transverse folds, but there is really nothing in the representation of the "button-hole" ulcers to differentiate this from other forms of colitis.

Double contouring of the evacuation picture which is evoked by production of mucus, as well as by ulceration, is sometimes also observed in the descending colon. In the most advanced stage the filling picture shows absence of haustration and saw-edge indentations (as in diverticulosis). Then the mucosal relief pattern is no longer recognizable. Sometimes a finely granular pattern may be recognized, which signifies pseudopolypoid formation. The sudden change from the abnormal to the normal relief picture in sharply defined sections of the large intestine is held to be characteristic of intestinal amoebiasis.

Any definite ruling regarding the exact localization of the process is difficult to formulate, but in the great majority of cases it is localized in the descending and sigmoid colons and, not infrequently, in the caecum and ascending colons; seldom in the transverse.

It is emphasized that radioscopy alone cannot be considered as of prime diagnostic importance, but as an accessory to faecal examination. It constitutes, however, a very definite indication for treatment. Thus when lesions of the caecum and ascending colon are present the administration of oral yatren is indicated.

The appendix may be involved and this has a definite bearing upon possible operation. One patient is cited with tenderness in the right iliac fossa. Definite lesions in the appendix were localized by radioscopy and the condition yielded altogether to energetic antiamoebic treatment. Radioscopy may also in case of necessity serve as an indication when operation should be performed.

In liver abscess the size of the liver shadow is an indication of the abscess, but does not serve to localize it exactly.

The localization of cancerous metastases in the liver has been rendered possible with Jodsol. The second attempt at outlining liver abscess with this substance was successful. An air vesicle was apparent in the liver substance, which subsequently was shown to be a portion of a loculated intrahepatic abscess.

The technique of Jodsol administration consists of preliminary testing of iodine toleration by means of 0·5 gm. potassium iodide. If negative, a plain X-ray of the abdominal cavity is taken and Jodsol is injected intravenously in doses of 250-300 mgm. of iodine per kilo. in 20 per cent. solution. The injection must be made slowly, 3 cc. per minute. After an interval of one hour the liver radiographs are taken.

The excellent radioscopic pictures which illustrate this paper are beyond criticism, but this must be directed towards their interpretation. To the reviewer it would appear that none of the intestinal appearances shown are peculiar to intestinal amoebiasis—rather he would suggest that either the primary microscopic diagnosis was at fault, or that there were superadded complications in some cases, such as polyposis (figs. 7 and 8) or diverticulosis (figs. 3 and 4), whilst two (figs. 5 and 6) are suggestive of a chronic inflammatory colitis, such as ulcerative colitis or chronic bacillary dysentery.] *P. Manson-Bahr.*

Bautista Serra (Juan). Contribución al estudio de las reacciones cutaneas en la amebiasis intestinal crónica. [**Cutaneous Reactions in Chronic Intestinal Amoebiasis.**]—*Rev. Argentina de Dermatosifilologia.* 1942. Vol. 26. Pt. 2. pp. 327–333.

The conditions referred to here are of an allergic nature and are to be distinguished entirely from amoebic lesions of the skin—cutaneous amoebiasis—such as may be found, for example, in the area surrounding a draining amoebic abscess. The former, to which the term *amoebides* has been given, are characterized by their being, as stated above, a manifestation of allergy, by absence of the amoeba from the skin lesion, by its presence in the faeces, and by their curability with anti-amoebic treatment. Associated symptoms are local pruritus, obstinate urticaria, perhaps with recurrence of dysentery. Brief notes are given of fifteen such cases, in which the patients would suffer from headache, constipation, disturbed sleep, nightmares, etc., together with some cutaneous manifestation such as a local pruritus, urticaria, eczema or pityriasis, which resisted all the usual forms of treatment, but cleared up when the discovery of *E. histolytica* was made (or other Entamoeba, *Entamoeba* [? *Endolimax*] *nana* in two cases) and the appropriate treatment adopted. [Wenyon (*Protozoology*, 1926, p. 240) states that there is no evidence that *Endolimax nana* is in any way pathogenic. This being so it is difficult to see how it can be responsible for cutaneous reactions.]

One very instructive example is that of a man employed in a cigar factory ; he had lesions of both hands and arms ascribed to his occupation and was treated ineffectually for some months till it was found that the stools contained *E. histolytica*. A course of emetine, followed by amoebarsone and enterovioform wrought complete cure.

H. Harold Scott.

RELAPSING FEVER.

COOPER (Eric L.). **Relapsing Fever in Tobruk.**—*Med. Jl. Australia.* 1942. June 6. 29th Year. Vol. 1. No. 23. pp. 635–637.

A description of 68 cases of relapsing fever which passed through an Australian General Hospital in Tobruk during May to August 1941.

Thirty-nine of the soldiers affected had been living in caves or in old dugouts constructed by the Italians, but these had not been occupied by either Italians or Libyans for the three months before the first appearance of the disease. Caves, dugouts, tank traps and trenches were infested with rodents. Ticks were found in slit trenches and a hospital orderly who had lived in a new trench developed relapsing fever. The disease also occurred in men from units living in various quarters scattered through the fortress area.

Nineteen men gave definite histories of tick bite. It is also stated that in a few cases the first pyrexial attack occurred 8 days after ticks had been removed from the skin. The number of attacks varied from 1 to 10, the mean being four. The total duration of illness before returning to duty varied from 3 to 26 weeks, the mean being 12 weeks.

The clinical aspects of the disease are reviewed; fever associated with the presence of spirochaetes in the blood was the only feature common to all these cases. Some patients had symptoms and signs suggesting involvement of the central nervous system, the reticulo-endothelial tissues, the renal tract, or the lungs; late involvement of the central nervous system with facial paralysis was relatively common. It is emphasized that the blood must be examined repeatedly, in thick films, in order to be sure of diagnosis, and that spirochaetes are most likely to be found at the beginning of a febrile attack.

The disease did not respond to arsenic given intravenously in the doses generally used for the treatment for louse-borne relapsing fever. Relapses occurred after arsenic treatment in 41 out of 57 patients, but the quantities of arsenic used in these patients were much less than those used in syphilis, so that a full opinion as to its efficacy cannot be given. [The species of tick is not stated and although the disease is presumably one of the North African rodent strains transmitted by various species of *Ornithodorus*, the statement that in many cases ticks were removed from the skin suggests that other genera may have been involved.] *E. Hindle.*

ROBINSON (P.). **Relapsing Fever in Addis Ababa.**—*Brit. Med. Jl.* 1942. Aug. 22. pp. 216–217.

A report based on 340 cases of relapsing fever [*S. recurrentis*] in Addis Ababa observed during the second half of 1941.

The infection is assumed to be transmitted by the body louse since mixed infections of relapsing fever and louse-borne typhus were common; spirochaetes were often found in crushed lice collected from relapsing fever patients; and ticks were not found nor any evidence of tick bites.

The routine method of diagnosis was the examination of thick blood films which revealed spirochaetes in most cases, even in the afebrile stage. Jaundice was frequent. A rash with a typical violet shade usually developed on the shoulders. Tongue pain, atrophy and

ulcerations were also typical. Systolic and diastolic murmurs were frequent and also additional evidence suggests that the disease causes cardiac lesions.

The author found that the serum of these patients agglutinated the Kingsbury strain of *Proteus* in a high titre, sometimes up to 1 : 3,200. This *XK* agglutination was found in 98 per cent. of the 168 cases examined and is considered to be the result of the relapsing fever infection.

The cases were treated by one, two or three injections of N.A.B., but relapses occurred. The haemorrhages caused by prothrombin deficiency were successfully treated with vitamin K, which is recommended for all relapsing fever cases.

The mortality rate was 3·5 per cent. Perisplenitis, spleen infarcts, and endocarditis were observed at necropsy. *E. Hindle.*

ROBINSON (G. G.). **The Quantitative Interaction of Spray Fluid and Active Principle in Determining the Toxicity of a Pyrethrum Preparation to the Argasid Tick,** *Ornithodorus moubata* **Murray.**—*Ann. Applied Biol.* 1942. Aug. Vol. 29. No. 3. pp. 290–300. With 3 figs. [14 refs.]

This paper forms part of a study aiming at the discovery of a contact insecticide suitable for use against *Ornithodorus moubata*. The author deals here chiefly with the results of applying an extract of Pyrethrum in white oil (initial B.P. : 325°C. ; viscosity : Redwood I at 20°C. 141 secs.) either directly to the tick or as a " film " on a surface over which the tick subsequently crawls. He shows that by either method, if the same total weight of pyrethrins is applied over a given area, these are more lethal in dilute solution (*e.g.*, 0·13 per cent.) when the total weight of the pyrethrins is small ; they are more lethal in concentrated solution (*e.g.*, 1·3 per cent.) if the total weight of pyrethrins is large. These results are explained by the extent to which the permeable areas of the cuticle are covered by insecticide in the two cases ; that is, " on a balance between the inherent toxicity of the active principle and the bulk or availability of the dose."

V. B. Wigglesworth.

LEPTOSPIROSIS.

SCHÜFFNER (W.). Die Bedeutung der Weilschen Krankheit bzw. der Leptospireninfektionen für Afrika. [**The Importance of Weil's Disease and other Leptospiral Infections in Africa.**]—*Deut. Trop. Ztschr.* 1941. May 1. Vol. 45. No. 9. pp. 257–272. With 19 figs.

A general account of the subject containing nothing original. [The main interest of this and other articles in this journal lies in their political implications as they are concerned with diseases in Africa. In the discussion following Schüffner's address, presumably given early in 1941, Uhlenhuth recommended the immediate organization of an Expedition to inspect their future place of work (" zukünftiges Arbeitsgebiet ") !] *E. Hindle.*

STARBUCK (Elizabeth B.) & WARD (Thomas G.). **Comparison of a Recently Developed Macroscopic Agglutination Test for the Diagnosis of Leptospiral Jaundice with the Standard Microscopic Test.**—*Jl. Infect. Dis.* 1942. Jan.–Feb. Vol. 70. No. 1. pp. 88–91. [Summary appears also in *Bulletin of Hygiene.*]

It will be readily acknowledged that a macrosopic test in which preserved leptospira culture could be used would be of the greatest value in diagnosis of leptospirosis. For the *microscopic* test the authors use cultures obtained on a modified Noguchi semi-solid medium, by incubating at 32°C. for 5–7 days; 0·2 cc. of such a culture of actively motile leptospira is mixed with an equal quantity of serum (diluted), and examined by dark-ground illumination for agglutination after being kept in a water-bath for 1½ hours at 37°C. The highest dilution causing agglutination gives the titre of the serum.

For the *macroscopic* test a formolized culture is used, a small amount of gentian violet being added to facilitate reading of results. One drop of the antigen and one of the various dilutions of serum are mixed in squares ruled on a slide, rocked for 10 minutes, and then examined over a white background by diffuse transmitted light. If the result is positive light blue aggregates are seen.

Three hundred and fifty-six sera were tested and the results are presented in the following table:—

Comparison of Results obtained by testing Sera for Agglutinins against L. icterohaemorrhagiae *by the Standard Microscopic and by the Macroscopic Test.*

Groups Tested	Number Tested	Microscopic Test		Macroscopic Test	
		Number Positive	Number Negative	Number Positive	Number Negative
University students ...	50	0	50	0	50
Specimens from Wassermann laboratory ...	294	9	285	2	292
Eagle test negative ...	253	9	244	2	251
Eagle test positive ...	41	0	41	0	41
Suspected cases of Weil's disease	7	3	4	3	4
Known positive sera ...	5	5	0	4	1
Total	356	17	339	9	347

Important points are that of 7 suspected cases of Weil's disease 3 were positive to both tests, microscopic and macroscopic. Of 5 positive to the former 4 were positive to the latter, the fifth being positive microscopically in low dilution only. The macroscopic test proved to be highly specific and no false positives were given by it, judging by the standard microscopic method.

Tests to compare the relative sensitivity of the two methods showed that the macroscopic was less sensitive than the microscopic. Of 24 human sera tested by the authors by both methods, 15 with a titre of above 1 : 200 by the microscopic were also positive by the macroscopic test; of 9 with titres of 1 : 100 or less by the former 8 were negative by the latter. The titre by the latter (macroscopic)

method was always much lower than that by the former (microscopic), but since patients with leptospiral jaundice usually give agglutination with their sera in very high dilution this lower degree of sensitivity is sufficient for diagnosis. Also it is more easily carried out and takes much less time than the microscopic test. *H. Harold Scott.*

YAWS.

Dupont (Adolphe) & Dubois (A.). Contribution à l'histopathologie du pian. [**The Histopathology of Yaws.**]—*Ann. Soc. Belge de Méd. Trop.* 1940. Dec. 31. Vol. 20. No. 4. pp. 461–477. With 8 figs. & 2 photos on 5 plates.

This article is difficult to abstract satisfactorily, for it describes minutely the histological changes observable in the cutaneous lesions of the different stages of yaws and compares them with those of syphilis and of leprosy. The chief of these differences will be referred to here; those wishing for more detailed information should consult the original where the lesions are well shown by a series of photomicrographs. The descriptions are based on biopsies from one patient with a primary yaw, four in the secondary and twenty in the later stages of the disease. Search for the spirochaete has not been attempted, for the portions of tissue excised were fixed in Bouin's fluid, which cannot be followed by silver staining. "The Congolese distinguish with confidence yaws from syphilis. . . . Moreover, syphilis provokes repeated abortion and affects infants born alive, whereas yaws has no effect on pregnancy and does not harm the foetus."

The primary lesion is a papule involving the epidermis and dermis, its centre undergoing necrosis leading to the production of an ulcer; cellular infiltration is limited even when the involvement of the epidermis is considerable. The papillae are very congested, the vessels dilated, and there is marked local oedema, the fluid containing polymorphonuclears, lymphocytes and histiocytes. The epidermal ridges are thickened, less at the periphery, more towards the centre. The polymorphonuclear cells penetrate the "mucous bodies of the dermal papillae" [these are not further defined] and the canals formed are "invaded by the oedema." The noteworthy point is that the ulceration attacks only those parts of the epithelium which cover the dermal papillae. In contradistinction to this the syphilitic chancre erodes the epidermis, setting up practically no reaction; it also extends more deeply.

In the second stage—the classic framboesia—the above changes are much more in evidence. There are large papules formed by inflammatory infiltration, always superficial and very compact, with marked hypertrophy of the epidermal ridges which are almost of a vegetating character. As the cellular infiltration increases, imbibition of oedema fluid by the connective tissue diminishes and the vascular congestion is less.

The dermal lesions of secondary syphilis often bear a close resemblance to those of yaws. In both there are: exuberant proliferation of the epithelium, dense infiltration with the plasmocyte as the predominant cell and invasion of the surface layers by polymorphonuclears with formation of a crust. In yaws, however, the inflammatory infiltration

is, as it were, condensed ; it barely extends beyond the limits of the dermal papilla and appears as an almost horizontal layer, sending out thin extensions into the corium. In a syphilitic condyloma the infiltration is more extensive, is usually prolonged into the corium, ensheathing its blood-vessels.

In the third, or gummatous, stage, the appearance is that of a large tuberculous follicle. The central part, which makes up nine-tenths of the mass, is formed of a dense homogeneous aggregation of epithelioid cells, with giant cells of the Langhans type, isolated or grouped ; here and there these cells undergo more or less complete caseation. In the midst of the mass one may see a number of quite healthy capillaries. Lymphocytes and plasmocytes encircle the central epithelioid mass. The most striking feature of the " yaws gumma " is the predominance, almost the exclusiveness, of the epithelioid element in the infiltration ; the common inflammation cells—lymphocytes and plasmocytes—play but a minor rôle. This is in marked distinction from the syphilitic gumma in which at the onset the plasmocyte, in later stages the lymphocyte, dominates the picture. In the latter also vascular lesions are important, the vessel walls are destroyed till there remains merely the elastic skeleton and a sclerosed mass, whereas in the former the epithelioid cells agglomerate and infiltrate between the structures of the vessel wall without destroying it, and little by little block the lumen.

Confusion with leprosy arises from similarity of the nodular infiltrations. Both show masses of epithelioid cells with scattered giant cells in greater or less abundance. In leprosy, however, the trails of inflammatory cells surround the branches of the cutaneous vessels and nerves, from the papillae to the subdermal *stratum retiforme* ; the epithelium itself largely escapes attack, and infiltration in the nerve filaments is characteristic. The infiltration involves both deep and superficial layers.

In yaws, on the contrary, the lesions are agglomerated. The superficial papules of the skin show an infiltration in the dermal papillae and the upper part of the corium in immediate contact with the epidermis, and form a large nodule with a halo of aggregated inflammatory cells more and more discrete and wider apart as the distance increases from the centre of the lesion. The inflammatory cells encroach on the epithelial layers, which they separate by penetration. In short, in both the infiltration is frankly tuberculoid ; in leprosy it has a tendency to spread in depth as well as superficially, whereas in yaws it remains superficial with a tendency to compact nodule formation. [See also this *Bulletin*, 1939, Vol. 36, p. 45.] *H. Harold Scott.*

LEPROSY.

DAVEY (T. F.). **Leprosy Control in the Owerri Province, Southern Nigeria.**—*Leprosy Review.* 1942. Apr.–July. Vol. 13. No. 2. pp. 31–46. With 2 maps.

This report records remarkable progress in the control of leprosy in a badly infected portion of Southern Nigeria. At the end of 1941, 1,072 cases, many requiring hospital treatment, were isolated in the central Uzuakoli settlement ; 2,000 more infective cases are voluntarily

isolated in model leper villages constructed on sanitary lines by the patients under the instruction of the staff of the settlement on sites provided free by the local Chiefs, with land to cultivate for their own maintenance. These and the other cases of leprosy in the surrounding villages were being treated at 29 clinics (with four more about to be provided) in which 7,183 cases are cared for, mostly early amenable ones, which are six to ten times as numerous as the advanced, all of whom live within five miles of the clinic they attend. In considerable areas complete control of all the leprosy cases has been attained, first by starting a clinic for treatment, and then, after the confidence of the villagers has been obtained, conducting a house-to-house survey, which leads to about three times as many cases being found and treated. Over 70 male nurses have been trained in the settlement by nursing sisters, together with a few Toc H workers and a number of educated native leprosy inspectors, who carry out the surveys and superintend the construction of the model leper villages, in which none but lepers are allowed to reside. Recorded infection rates vary, in different villages, from 1·3 to 15·2 per cent. By these very economical measures the only limit to the extension of the work is the provision of more workers and moderate funds, for at present only two medical men superintend the whole of the work in caring for some 10,000 leprosy cases on the books. *L. Rogers.*

Bloss (J. F. E.). **Li Rangu Leper Settlement.**—*Report of Sudan Med. Service for Year 1940.* pp. 16–21.

The estimated number of leprosy cases in the Sudan is about 8,000 ; the known cases number 5,888. Of the latter 3,637 are in the humid southern Equatoria province and 1,992 more are in the Central Kordofan area, leaving only 259 in the dry northern area. The largest number are in the Li Rangu district where 956 remain in the settlement and 1,458 are out-patients in the Yambio sub-district ; the latter obtain treatment at dispensaries. The leprosy incidence here is at the high rate of 4 per cent. From Li Rangu 299 patients were discharged in 1940 and a further 120 were to be set free in the next year ; most of the remaining cases will be those which are bacterially positive. Pure *Hydnocarpus wightiana* oil is used for injection treatment. Complications due to bilharzia and ankylostomiasis have been greatly reduced by treatment. *L. Rogers.*

Leprosy in India. 1942. Apr. Vol. 14. No. 2. pp. 41–72.—**Report of the Leprosy Survey Sub-Committee of the Indian Research Fund Association, September, 1941.**

Under the chairmanship of Dr. J. Lowe this committee dealt with both the principles and practical details of leprosy surveys of three types in India. (1) Surveys of large areas to ascertain the approximate incidence, distribution and epidemiological features where little is known about the disease. Selected portions of the district or province are carefully studied as samples of the whole, including parts believed to have high and low incidence. The examination of contacts of known cases and of all village children reveals unreported cases. Schedules are given for entry of the results obtained on uniform lines. (2) Surveys of smaller areas with a view to the detection and recording of as many

as possible of the cases where the disease is already known to be prevalent, with full examination of the population of a union or group of villages for five miles around an anti-leprosy centre. (3) Specially detailed studies of the epidemiology of leprosy, with examination of every individual village by village, of small populations of three to ten thousand people carried out by experienced workers. Detailed records with all the cases marked on village maps are prepared to allow of re-surveys to ascertain the changes taking place within a given time. The types, age incidence, type distribution in relation to age, evidence of increase or decrease are all noted to allow of the relative importance of leprosy as a serious public health problem to be ascertained. Where the disease is prevalent anti-leprosy work includes the provision for isolation of infective cases in homes, villages, colonies or institutions, and provision for diagnosis and treatment at existing hospitals, dispensaries and in special leprosy clinics. Propaganda is essential to create, in the villages, public opinion that will insist on isolation of the infective cases, to be established and maintained. Re-surveys are of great value in estimating the progress being made in any area.

L. Rogers.

MUIR (E.). **Report of Leprosy in British Guiana.**—*Leprosy Review.* 1942. Apr.–July. Vol. 13. No. 2. pp. 22–31.

This report brings out well the success of the 15 years' devoted work of Dr. F. G. ROSE. British Guiana was the first of our colonies to modify, at the suggestion of B.E.L.R.A., their compulsory segregation laws relating to all types and forms of leprosy, so as to permit early and uninfective cases to be treated at out-patient leprosy clinics. There are now nine clinics treating 500 cases out of a total of 1,000. The 500 more advanced and infective patients are cared for in the up-to-date leprosy colony at Mahaica, most of the inmates of which are now voluntary admissions for the sake of treatment. Yet in 1923, before the law was relaxed, only 267 cases were segregated at Mahaica, but by 1932, 747 lepers were in residence there. The value of regular and persistent treatment is shown by the fact that among the patients who received 61 to 100 per cent. of the prescribed course, 71·4 per cent. became "arrested cases," against only 16·7 per cent. of those who received 60 per cent. or less of the course. Child cases are accommodated in a separate building and healthy children of leprosy patients in the Lady Denham Home. During his 1941 visit Muir records that he saw many formerly advanced lepromatous patients who had been free from infection and active symptoms for years; he concluded that "There is good reason to believe that this decline in notifications [namely of the yearly admissions from 40–100 to only 39] is the result of actual decrease of leprosy in the colony."

L. Rogers.

FAGET (G. H.). **The Story of the National Leprosarium (U.S. Marine Hospital), Carville, Louisiana.**—*Public Health Rep.* 1942. May 1. Vol. 57. No. 18. pp. 641–652. With 4 figs. on 2 plates.

This is an interesting account of the best equipped leprosarium in the world. The author first refers to the unjustified fear of leprosy among the general public. The origin and spread of leprosy in the United States is attributed to its introduction into Louisiana by the Acadians from Canada, by Scandinavians into Minnesota, and by negro

slaves and Chinese and other immigrants. The establishment of the Louisiana leper settlement and its conversion into the present federal settlement at Carville in 1921 is described. Extensions at a cost of $645,000 enlarged the accommodation from 90 to 425 by 1923, and in 1940 the wooden buildings were replaced by permanent structures at a cost of approximately $2,500,000. Many further expensive additions include electricity and water supplies, laundries, departments for radiology, dentistry, bacteriology, physiotherapy, etc., and a recreation building costing $140,000. The medical staff includes five doctors, one dentist and three consultants, and a full-time bacteriologist. Post-graduate teaching is carried out.

During the earlier period of State control 338 patients were admitted, all but 16 from Louisiana State. Between 1921 and 1942, 1,034 were admitted, 593 died, 53 left for foreign countries and 309 have been discharged as no longer a menace to public health; 58 of these have returned with relapses. Of the total patients admitted 404 were foreign born, Louisiana furnished 576, California 194, Texas 192, New York 118, and Florida 76, leaving only 215 from all the other States. In treatment vitamins were used as tonics, but vitamin A in the form of " alfon " did not seem to be as effective as had been hoped. Vitamin B_1 was of value in relieving painful leprous neuritis, but diphtheria toxoid in a carefully controlled trial was as disappointing as elsewhere. *L. Rogers.*

DE SOUZA-ARAUJO (H. C.). Infecção de ratos brancos com suco ganglionar de leproso, seguida do isolamento dum bacilo acido-alcool resistente de orgãos do murideo, em meio de Loewenstein. Nota prévia. [**Infection of White Rats with Gland-Juice from a Leper; Isolation and Growth of an Acid-fast Organism.**]—*Mem. Inst. Oswaldo Cruz.* 1941. Nov. Vol. 36. No. 3. pp. 379–385. With 15 figs. on 4 plates.

Though this is a short paper, in fact a preliminary note, its importance calls for a full account. In view of repeated fruitless attempts to cultivate Hansen's bacillus from emulsions of lepromata, skin lesions, nasal mucus, and blood, the author started a series of investigations on the sputum and gland-juice of lepers. From 42 samples of sputum he obtained 11 cultures of Mycobacteria, 10 of which had all the characteristics of the human, eugonic type of *M. tuberculosis*. Of 15 samples of gland-juice seeded on media appropriate for growing acid-fast bacilli, one gave a pure culture which also had the characteristics of *Myco. tuberculosis*. He then proceeded on the following lines, suggested by the work of MARCHOUX.

A man of 26 years, showing typical symptoms of leprosy—thickening of the ears, enlargement of the cervical and inguinal glands, swollen nose, macules on the forehead, cheeks, chest and abdomen, lepromata on buttocks, thigh and elsewhere—came under the author's observation in August 1938. The nasal mucus, fluid from skin puncture, juice from a cervical gland, all showed Hansen's organism; the Witebsky reaction was 6 plus, the Wassermann reaction negative. During 1939 the patient suffered from repeated leprotic reactions with exacerbation of the cutaneous lesions and suppuration of the groin glands, the pus containing abundant bacilli. In June 1939, some gland juice, rich in bacilli, was emulsified in saline and injected (as suggested by Marchoux for Stéfansky's organism) subcutaneously in the axilla of a white rat.

Five months later the animal was killed; the axillary and inguinal glands were much enlarged and contained many bacilli like those of Hansen. Smears of the viscera did not show them; probably it was too early for visceral involvement. In January 1940, pus from the patient's groin gland was inoculated into three white rats subcutaneously in the axilla and the following month gland-juice into a fourth rat. (Frei's reaction was tried because of the inguinal adenitis, but proved negative, thus ruling out lymphogranuloma inguinale). The sputum contained no tubercle bacilli and guineapig inoculation was also negative.

Fifteen months later one of the inoculated rats died, showing areas of alopecia on the back and lesions, containing bacilli, in the spleen. Two months later another died and autopsy revealed glands and viscera rich in acid-fast bacilli. These organs were sent to Dr. Margarinos Torres, together with a portion of the liver on ice, for seeding. This was triturated and sown on Löwenstein's medium. Dr. Torres reported that the tissues showed generalized infection with lepromatous lesions. A detailed description is given and illustrated by photomicrographs. A fortnight after the sowing the medium showed growth of acid-fast organisms, in bundles and clumps. Four rats and a monkey were inoculated with the culture on the 2nd and 11th August, 1941, respectively [the results of these will doubtless be reported in due course].

Eighteen months after being inoculated the third rat was killed; it showed alopecia on the back, the root of the tail and the right hip; the glands were enlarged and contained what seemed to be purely Hansen's bacilli, but smears of the viscera did not show any.

The author concludes:—

1. The gland juice of a leper is infective for white rats.
2. After an incubation period of 15–18 months, rats inoculated with this gland-juice show typical signs of leprosy in their glands and viscera.
3. Seeding, on Löwenstein's medium, of emulsions of the organs of infected rats gives rise to the growth of a pure culture of an acid-fast bacillus which is not *Myco. tuberculosis*.
4. This work ought to be repeated on a larger scale, as it points out a way of clearing up obscurities in the aetiology of human leprosy.

H. Harold Scott.

MARCHOUX (E.), CHORINE (V.), CHABAUD (A.) & TISSEUIL (J.). Essais négatifs de la transmission de la lèpre humaine au hamster de Syrie, *Cricetus auratus*. [**Negative Leprosy Transmission Trials on Hamsters.**]—*Ann. Inst. Pasteur.* 1942. Feb. Vol. 68. No. 2. pp. 99–105.

This paper records the negative results of attempts, by different methods of inoculation, to infect hamsters with human leprosy material. The experiments were carried out with 40 animals, who had had their spleens removed; these, with 25 in which the spleens were intact, were inoculated subcutaneously with emulsions or small fragments of lepromas; five others with intact spleens were inoculated intraperitoneally. The authors point out that the long survival of lepra bacilli in large numbers at the site of injection, or in the internal organs in small numbers, furnishes no proof of infection, because ARAUJO in Brazil showed that this may occur after inoculation of killed lepra bacilli. They obtained no evidence of multiplication of the bacilli

in any of the inoculated animals. The hope that infected hamsters might be used for chemotherapeutic tests must therefore be abandoned. *L. Rogers.*

DE SOUZA-ARAUJO (H. C.). Poderá o carrapato transmitir a lepra? [**Can Leprosy be transmitted by Ticks?**]—*Mem. Inst. Oswaldo Cruz.* 1941. Dec., Vol. 36. No. 4. pp. 577–585. With 2 plates (1 coloured). [10 refs.] English summary.

"1. RUDOLPH (1918) stated that until 13 days after the last feeding on lepers nymphs of *Amblyomma cajennense* had in their intestines leprosy bacilli, which he considered as alive. The author of the present paper found that such bacilli are in degeneration after a week of the sucking, perhaps because they are being digested.

"2. The examined nymphs, within the first 48 hours after sucking on active lepers, showed in smears of their sediment, in more than 60 per cent. of them, homogeneous bacilli, well stained by Ziehl-Neelsen method, in small bundles or in small spheric or ovoid globies, with the characteristics of Hansen's bacillus. By culture in Loewenstein medium of such material and by inoculation of it in white rats the author supposes that it is possible to prove the vitality and virulence of the organisms.

"3. If the *Amblyomma* ticks change their hosts during their three suckings of blood, necessary to their life cycle, such ticks may be occasional carriers of leprosy bacilli.

"4. Considering that ticks are widespread in the States of Brazil and that leprosy is an endemo-epidemic scourge in rural zones of many States, it is urgent to determine the real rôle of such parasites in the transmission of human leprosy.

"*Addendum*: After 62 days' incubation at 37°C. the author obtained a pure culture of an acid-fast bacillus from sediment of ticks captured on lepers.

"The medium used was that of Loewenstein."

CHABAUD (A.). Altération du bacille de Hansen par les fixateurs: Rôle protecteur de l'acide phénique. [**Alteration of Hansen's Bacillus by Fixation Agents.**]—*Ann. Inst. Pasteur.* 1942. Feb. Vol. 68. No. 2. pp. 106–113.

The author in this note points out that long fixation of human leprosy material in Bouin alcohol alters the bacilli so that after about three weeks or more the lepra bacilli are no longer stained by the Ziehl method. This change seems to depend on the action of formol, salts of the heavy metals and especially on the acidity of the fixation agent. Carbolic acid retards this alteration of the bacilli so that they can be stained up to 136 days in a carbolic fixation agent.

[Bouin solution contains 1 gm. picric acid in 150 cc. of 80 per cent. alcohol, 60 cc. commercial formalin, 15 cc. glacial acetic acid. The carbolic fixation fluid contains 60 cc. of 80 per cent. alcohol, 15 gm. phenol, 5 cc. commercial formalin, 2 cc. acetic acid.] *L. Rogers.*

DHARMENDRA. **The Immunological Skin Tests in Leprosy. Part I. The Isolation of a Protein Antigen of** *Mycobacterium leprae.*—*Indian Jl. Med. Res.* 1942. Jan. Vol. 30. No. 1. pp. 1–7.

"1. The main features of the lepromin test (the Mitsuda reaction) are outlined. It is considered that the delay in the appearance of the

classical nodular reaction, and possibly the non-specific nature of the reaction may be caused by the nature of the material injected. The need for a refined antigen is stressed.

" 2. A method for obtaining from excised nodules leprosy bacilli free from tissue is described. Bacilli have been found to be the active constituents of lepromin.

" 3. The bacilli were ground for several hours in an agate mortar and were fractionated into a saline-soluble portion and an insoluble residue. From the soluble portion, protein and polysaccharide have been obtained ; from the insoluble residue, various lipoid fractions have been separated.

" 4. Tests with the different fractions of the bacilli have shown that, of all the fractions isolated, only the protein is definitely antigenic and that it produces only an early reaction.

" 5. By extracting the ground bacilli with different solvents, three protein fractions—acid-soluble protein, nucleo-protein and alcohol-soluble protein—have been isolated. All the three fractions give rise to early reactions in the neural cases of leprosy."

DHARMENDRA & LOWE (J.). **The Immunological Skin Tests in Leprosy. Part II. The Isolated Protein Antigen in Relation to the Classical Mitsuda Reaction and the Early Reaction to Lepromin.**—*Indian Jl. Med. Res.* 1942. Jan. Vol. 30. No. 1. pp. 9–15. [11 refs.]

" 1. The intradermal injections of preparations from leprous material are capable of causing reactions of three different clinical types : The classical Mitsuda reaction (nodular), the early erythematous reaction preceding the classical reaction, and the erythematous reaction followed by no late reaction.

" 2. Since protein is the only definitely antigenic fraction of the *Myco. leprae* and since all the different types of reaction can be explained on the basis of this one antigen, it is believed they are actually caused by it. If at the time of injection all the antigen is free to act at once, only an early reaction is produced. If only part of the antigen is free, both early and late reactions are produced, the early reaction by the free antigen and the late by the same antigen which is liberated slowly from breaking down of the injected bacilli. If none of the antigen is free a late reaction only will be produced.

" 3. Since late reaction is not produced either by any of the isolated fractions or by the final residue of the bacilli, the idea that the early and late reactions are caused by different antigens is disproved.

" Our work, however, shows the presence of more than one antigen of protein nature.

" 4. When compared to the classical Mitsuda test, the test with the isolated antigen is found to be at least as sensitive, to give results of the same significance and to have great advantages, particularly rapid results and the absence of unpleasant nodules and ulcers. It is, therefore, suggested that, for performing skin tests in leprosy, the isolated antigen may be used in place of ordinary lepromin.

" 5. By providing an explanation for the lateness of the reaction, the observations reported herein have brought the Mitsuda reaction more in line with the allergic skin tests. The lack of specificity and the negative results in cases of the ' lepromatous ' type have still to be explained before the reaction can be admitted as one of specific allergy. Work with the isolated antigen will facilitate a study of these phenomena."

DHARMENDRA & LOWE (J.). **The Immunological Skin Test in Leprosy. Part III. The Isolated Protein Antigen in Relation to the Antigens used by other Workers.**—*Indian Jl. Med. Res.* 1942. Jan. Vol. 30. No. 1. pp. 17–22. [14 refs.]

In this short paper the authors discuss antigens used by other workers in the elucidation of the Mitsuda test. That of NAGAI (1938) was made by keeping leprous nodules for long periods in 5 to 10 per cent. lecithin or boiling for half an hour in the same solution to produce loss of acid-fastness and degeneration into granules. Intradermal injections produced similar reactions to those of ordinary lepromin. KITANO and INOUE (1941) broke down the lepra bacilli by physical means through the use of ultra-supersonic waves. Their lepromin produced stronger early but weaker late reactions than ordinary lepromin, very similar to those of the present authors. A few other workers have attempted to isolate the antigenic fraction from emulsions made by grinding up lepromatous tissue. The active non-lipoid fraction of VILLELA and his co-workers could not be a protein antigen and it is believed that their methods would neither break down the bacilli nor liberate the antigen. HENDERSON (1940) isolated proteins from leprous spleens rich in acid-fast bacilli by grinding dried spleen at −70°C. This produced only very slight early reactions of the "tuberculin" type. It thus resembles a weak antigen similar to that of the present workers. A proteose isolated from the brains of leprous patients by BERNY and MAUZE produced a different reaction from that of a bacillary antigen.

L. Rogers.

DUBOIS (A.). Notes pharmacologiques sur le buerre de Caloncoba. [**The Pharmacology of Caloncoba Butter.**]—*Ann. Soc. Belge de Méd. Trop.* 1940. Sept. 30. Vol. 20. No. 3. pp. 249–256.

PALDROK (A.). Ergebnisse meiner spezifischen Leprabehandlung in Estland in den letzten 20 Jahren. [**Results of the Author's Specific Treatment for Leprosy in Esthonia : Experience of 20 Years.**]—*Acta Med. Scandinavica.* 1941. Vol. 108. No. 5. pp. 374–383. [16 refs.]

BURNET (Et.) & CABASSO (V.). Action de différentes huiles sur le bacille de Stefansky. [**Action of Oils on the Bacillus of Stéfansky.**]—*Arch. Inst. Pasteur de Tunis.* 1941. Dec. Vol. 30. No. 3–4. pp. 203–226.

This paper deals with the action of oils on the structure and vitality of the rat leprosy bacillus. The authors use the terms reduced or residual for the granulation and segmentation of acid-fast bacilli, which may be observed to some extent in the living tissues, and more extensively in bacilli preserved outside the body. Stéfansky's bacillus of rat leprosy retains its vitality in saline up to about one month and its acid-fast rod-shaped form for months or even a year. If, however, it is suspended in paraffin oil, agglutination is seen in a few days and the organisms become granular and thin. Similar changes occur when it is suspended in oil of turpentine from pine trees and also in chaulmoogra oil *in vitro*, though more slowly. One or two black granules may also appear in them, and then tend to break up. Inoculation into rats shows that the organism has lost its vitality and power to infect the animals. The authors report the experimental inoculation of guineapigs and of rats with both large and small doses of such

" reduced " Stéfansky's bacillus subcutaneously, intraperitoneally and into the testis. The vitality of the bacilli is lost after 3 days in paraffin, 4–7 days in chaulmoogra, but they are living after 23 days in olive oil. When the bacilli, impregnated with paraffin or chaulmoogra oil, are injected in small doses the development of leprous lesions is prohibited, but months later acid-fast rod-shaped bacilli are found at the site of injection. After large doses, such as 5 milligrammes of the oil emulsion, the bacilli may be found in internal organs as well as locally, and the lesions resemble to some extent those produced by mixtures of tubercle bacilli with oils.

Attempts at treatment of infected mice by subcutaneous injections of living Stéfansky's bacillus, the instillation into the nose or inhalations of the vapour of pine oils, did not modify the course of the disease. Solutions of the bacillus in the oils, freed as far as possible from remaining bacilli by centrifuging, were injected to ascertain the effects of the injection of the soluble portions, but no lesions were produced. The experiments on the same lines with guineapigs showed that, in place of the very slight effects in rats, in the case of guineapigs Stéfansky's bacillus in oils produces much more marked effects similar to those with other acid-fast bacilli, such as tubercle and paratubercle organisms. The authors attribute this to the action of chemical substances common to these acid-fast organisms ; the lesions, therefore, cannot be regarded as strictly specific. *L. Rogers.*

HELMINTHIASIS.

Tucker (C. B.) & Chisolm (J. M.). **A Study of Intestinal Parasites in Relation to Excreta Disposal Facilities in Cocke County, Tennessee, 1940.**—*Southern Med. Jl.* 1942. May. Vol. 35. No. 5. pp. 476–483.

Faecal specimens were taken from 10·5 per cent. of the population of Cocke County, Tennessee, to determine the incidence of *Necator americanus, Ascaris lumbricoides, Trichuris trichiura* and *Hymenolepis nana*. This population sample corresponded closely to the sample taken from the same county by the Rockefeller Sanitary Commission in 1913. In 1940, 1·1 per cent. of the people examined (2,528 in all) had eggs of *Necator americanus*, a proportion 69 per cent. lower than that found in 1913. Probably the reduction was greater still, because in 1913 the simple smear method of examination was used, while in 1940 the Willis salt floatation method was used and, when *Necator americanus* was found, egg counts were done by the Stoll and Hausheer small drop dilution method ; the smear method probably missed a proportion of infestations. Infestation with *N. americanus* occurred 4·9 times as often in the mountainous eastern half of the county, where sandy-clay to sandy soils predominate, as in the non-mountainous western half, where clay soils predominate. In 1913 the prevalence of *N. americanus* was 2·5 times as great in the mountainous half. Thus there was a greater decrease in the non-mountainous area, which the authors ascribe to the differences in the soil and to greater improvements in education, sanitation and economic conditions in the non-mountainous area. Infestations with *N. americanus* high enough to

cause clinical symptoms (2,600 eggs per gm. laid down by KELLER *et al.* after a previous survey of the same county in 1930) occurred in 31 per cent. of the persons examined.

The incidence of *Ascaris lumbricoides* in 1940 was about half of that in 1913 (49·1 per cent. in 1913 and 24·3 per cent. in 1940). A reduction in the incidence of this species had occurred in both the mountainous and the non-mountainous areas. There had been little change, since 1913, in the incidence of *Trichuris trichiura* and *Hymenolepis nana*.

No account was taken of race, because the coloured population was too small to be significant. Of the persons examined 27 per cent. harboured one only of the four species recorded, 10 per cent. harboured two of them, 1 per cent. harboured three of them, and one or more of the four occurred in 38 per cent. Of the 2,528 persons examined 2 per cent. used sewers or septic tanks for excreta disposal, 32 per cent. used fly-proof privies, 34 per cent. had approved facilities, and 37 per cent. had no facilities at all. More had approved facilities in the non-mountainous areas. Among those who had no facilities *N. americanus* occurred 2·6 times as often, *A. lumbricoides* 2·4 times as often and *T. trichiura* 1·8 times as often as among persons with approved facilities.

In a discussion following this paper, Keller pointed out that the reduction of 69 per cent. in the incidence of *N. americanus* corresponded with the adjusted percentage reduction of 68·3 per cent. reported in 1940 from a study of six Southern States. He thought it was due, not to treatment, because no systematic treatment had been done since 1913, nor to excreta disposal, because the provision of facilities for this was too recent, but to a natural decline of hookworm in Cocke County. He quoted figures to show that more than a quarter of the white persons harbouring hookworm in the six Southern States referred to have a worm burden high enough to produce clinical symptoms (26·7 per cent. have 2,600 or more eggs per gm. of faeces), so that a hookworm control problem exists. He referred to the methods of survey and control suggested by ANDREWS [this *Bulletin*, 1942, Vol. 39, p. 772]. Dr. RICKS suggested that the Faust zinc sulphate centrifugal concentration method should be used instead of the Willis technique. He was not certain that it was better for hookworm, but it did reveal other parasites. TUCKER and CHISHOLM think that the Stoll and Hausheer method is not so good for detecting *N. americanus* as the Willis method, but that it is better than the Willis method for the detection of Ascaris eggs.

G. Lapage.

MOHR (Werner). Wurminfektionen bei den rückgekehrten Afrika-Deutschen. Beobachtungen im Tropeninstitut in den Jahren 1936–1940. [**Worm Infestations in Germans returning Home from Africa. Observations made in the Tropical Institute (in Hamburg) during the Years 1936 1940.** –*Deut. Trop. Ztschr.* 1941. May 15. Vol. 45. No. 10. pp. 307–315.

An examination in Hamburg of Germans, all of whom had lived a long time (often 10 years) in Africa, showed that diseases due to helminths were most important. The seamen among these patients were much less heavily infested than the others, having had less contact with the sources of infestation on land. Among the helminth infestations hookworm was the most important. The effects of these worms were more severe in the tropical areas, which favour the develop-

ment of these nematodes. When infestations were light, symptoms were often slight or absent ; other patients complained of occasional pains, mostly in the right epigastrium, and of abdominal discomfort. More severe infestations caused symptoms of anaemia. Severe infestations were rare and the severest form of the disease was never found. The relative absence of symptoms is explained by the blood examinations done on 61 out of 138 patients ; the haemoglobin percentage was 80–100 in 37 of these, 70–80 in 16, 60–70 in 6, and below 50 in 2, one of these latter having malaria also. Heavier infestations and more severe anaemia were found in patients returning from Brazil.

In children returning from Africa Mohr never found the devastating consequences of hookworm infestation seen in children returning from South America ; the latter were very backward, both mentally and physically, children seven years old being not much bigger or mentally more advanced than normal children three years old. Mohr ascribes this difference to the fact that German settlers in Brazil and South America get an insufficient living in tropical and subtropical climates, while Germans in Africa are mostly farmers better versed in rules of health. All other helminth infestations were less important, but could cause serious disease.

Taenia saginata was the next most common helminth found, occurring chiefly in Germans from East Africa, where, as WETZEL pointed out in the subsequent discussion of Mohr's paper, the cattle are heavily infested with this parasite. Out of 45 human infestations only three were in children or persons under 20. Only one patient had *T. solium* and one had *Hymenolepis nana*. An interesting detail is the fact that two of the patients had more than one tapeworm, two being present in each of them. Ascaris and Enterobius were not so common as tapeworms and occurred chiefly in children. Trichuris eggs were often found, but the infection did not cause any symptoms.

Schistosoma mansoni and *S. haematobium* were more important than the four worms just mentioned. Both were repeatedly diagnosed, but infestations were slight ; there was only one severe case, which came from North Africa ; all the others came from East Africa and Angola and they were comparatively light. *S. mansoni* occurred only in cases from E. Africa.

Filarial infestations were found in people from the Cameroons, Fernando Po and Spanish Guinea. *Acanthocheilonema perstans* seemed to preponderate.

A history of Calabar swellings (*Loa loa*) was found in one series of patients, and these showed a high eosinophilia without microfilariae in the blood ; occasionally Calabar swellings were still present. Complement fixation and skin tests were done, but these were not very specific, being positive also when high hookworm infestations were present.

Only one case of Guineaworm was seen, the patient being a native from the west coast of Africa ; the worms were encapsulated and calcified in the muscles of the back and scrotum and the X-ray diagnosis was confirmed by autopsy.

Eosinophilia was often found when no worms were present. Light infestations sometimes showed a high eosinophilia and heavy ones a low eosinophilia.

Infestations with Trichuris showed only a slight eosinophilia or none at all.

Treatment of hookworm infestation was carried out with ascaridol, which could cause urticaria and a high eosinophilia, possibly due to the release of increased amounts of toxin from the dead worms.

Hookworm infestations were also treated with oil of chenopodium, secretin or the combination of ascaridol and secretin called Bedermin. Enterobius infestations were treated with Lubisan, tapeworms were treated with extract of *Filix mas*, with atropin half an hour before it was given. For *Schistosoma mansoni* and *S. haematobium* fouadin was used, or intravenous injections of 1 per cent. solution of Tartarus stibiatus [tartar emetic] in doses of 6 cc. rising to 15 cc. with a total dose of not more than 1·3–1·5 gm. Filaria infestations were not treated. *G. Lapage.*

Davey (D. G.) & Innes (J. R. M.). **The Present Position of Phenothiazine as an Anthelmintic.**—*Vet. Bull.* 1942. Aug. Vol. 12. No. 8. pp. R7–R14. [97 refs.]

This article is a valuable summary of the experience so far gained in the use of phenothiazine in animals and man. It should be read in its entirety, but for readers of this *Bulletin* the following extracts are of main interest. Much of the work referred to has already been abstracted in this *Bulletin*, but it is useful to bring the experiences together.

Specific Anthelmintic Action of Phenothiazine.

" (8) *In Man.*—Manson-Bahr (1940) was the first to try phenothiazine as an anthelmintic in human beings. He used it in three patients with *Ancylostoma duodenale*, in nine patients with *Ascaris lumbricoides*, and in nine patients with *Enterobius vermicularis*. His results against the hookworm were entirely negative, even with a dose as high as 30–40 g. Against *Ascaris* some degree of positive action was obtained ; two patients, given 8 g. daily for three days followed by a dose of Glauber's salt, were completely freed of the worms, but from seven others, given doses of 16–48 g., only one round worm was recovered. Against *Enterobius* the results were very good, all the patients being cured. The doses advised against this worm were :—2 g. per day for seven days to children under eight years of age, half this dose for children under four, and 8 g. per day for five days to adults. We now know that these doses are too high to be generally recommended.

" Kuitunen-Ekbaum (1941) gave phenothiazine to 89 children and nine adults for the treatment of enterobiasis. The doses were 4–5 g. for children 2–5 years old, 6 g. for those 6–8 years old, and 8 g. for those nine years and over. A dose was apparently spread over 4–6 consecutive days, so that each child received 1 g. or less of the drug per day. It will be noticed that the dose for the first age group is about the same as that recommended by Manson-Bahr, but that for the second age group is, at the most, half of the Manson-Bahr dose, and that for the third group is only a fifth of it. The drug was administered to children in the food (in porridge or cereals) and to adults either in water or in capsules. No laxative was given. Of the 89 children and nine adults treated, 76 children and eight adults were cured by the first course of treatment ; the remainder, except for one child, responded to a second course. No toxic effects of the treatment were observed.

" Hubble (1941) also showed that phenothiazine, in a dose of 1 g. per 10 lb. body weight spread over 3–5 days, is an effective remedy against *Enterobius*, but it is a remedy which is " probably too dangerous . . . to be used in the routine treatment " of this worm. He makes this conclusion because, out of the 30 or so cases which he treated, three developed haemolytic anaemia. His warning seems to have been justified, for a case

has been reported (1942, *Lancet*, **242,** 86) in which a girl of six years died of acute haemolytic anaemia after taking phenothiazine. It had been advised to give the girl a ½ g. tablet four times a day; three tablets were taken on the first day, four on each of the next three days, and two on the fifth day—a total dosage of 8·5 g. She became ill on the fifth day with slight facial icterus and headache, and on the tenth day she was admitted to hospital with slight jaundice, a rapid pulse and a temperature of 101°F. A blood examination showed red cells 1,250,000, haemoglobin 26%, white cells 16,850, normoblasts 1%, reticulocytes 22·6%, platelets 236,000 per cu. mm. A drip transfusion of 10 oz. whole blood given the day after admission was followed by a rigor, a rise of temperature to 106·8°F. and a pulse rate of 160. She died about 12 hours after the transfusion.

"Elliot (1942) has recorded an interesting and successful treatment of dracontiasis with phenothiazine. A warm olive oil suspension of the drug is injected intramuscularly as near the course of the worm as possible. Two or three sites are injected with ½–1 g. of phenothiazine, and treatment may be repeated at weekly intervals. No toxic effects have been encountered even when as much as 8 g. (2 g. at weekly intervals) has been given. . . .

"*Toxic Effects in Human Beings.*—The earliest report of a toxic action of phenothiazine on human beings seems to be that of DeEds, Stockton and Thomas (1939), who were using the drug as a urinary antiseptic. Three of their patients, after receiving 19·9 g., 22·4 g., and 28·1 g., spread respectively over 8, 10, and 67 days, developed secondary anaemia. Hubble (1941) also showed that phenothiazine might produce haemolytic anaemia in children. He treated some 30 cases for enterobiasis and three of them developed anaemia and toxic hepatitis. Johnston (1942) described a case of toxic effect in a girl aged seven who was given 1 g. phenothiazine twice daily for five days. Treatment was followed by collapse, slight icterus, delirium, fever and increased pulse rate and anaemia. Blood transfusion was resorted to and the girl recovered. Finally, there is the fatal case already mentioned; after autopsy it was considered that "the appearance of the blood, spleen and marrow were consistent with a haemolytic anaemia . . . . Sections of liver, heart and kidneys showed no microscopical evidence of toxic damage," and there was "nothing to connect the blood transfusion with death." Thus eight patients out of a total of about 220 have exhibited toxic effects following treatment with phenothiazine.

"An interesting effect of the drug on men exposed to the sun, who were using a suspension of the substance as a spray in the control of codling moth, has been described by Newcomer (1937) and DeEds, Wilson and Thomas (1940). Some of the men complained of an intense itching, irritation and reddening of the skin, effects which sometimes approximated to those of severe sunburn. DeEds and his co-workers suggest that it is due, not to a local irritant action of phenothiazine, but to a photosensitization produced directly or indirectly by the presence of the oxidation-reduction system, thionol-leucothionol, in the tissues. The presence of this system in the tissues would be the result of the oxidation of phenothiazine taken in through the mouth."

C. W.

SCHNITZER (R. J.), SIEBENMANN (C.) & BETT (H. D.). **Chemical and Toxicological Studies on Phenothiazine.**—*Canadian Public Health Jl.* 1942. Jan. Vol. 33. No. 1. pp. 17–24. [16 refs.]

After briefly describing phenothiazine and its purification by recrystallization from toluene and benzene, the authors give what they regard as a better and cheaper method of purification by precipitation in the presence of a stabilizing reducing agent. A 20 per cent. solution

of commercial phenothiazine in cold acetone is treated with charcoal for 30 minutes and then filtered into twice its volume of 1 per cent. sodium formaldehyde sulphoxylate. The immediate microcrystalline precipitate must be dried completely (in a vacuum oven at 60°C.) or it will not be stable.

Phenothiazine thus purified was given as a suspension in gum acacia to white mice by the mouth to test its toxicity. After the administration of 5 gm. per kgm. mouse weight as a single dose or of 16 daily doses of 1·25 gm. per kgm. (20 gm. in all), there were no ill effects clinically or at autopsy. There was no anaemia.

Guineapigs did not tolerate the drug so well. Repeated daily doses of 2 gm. per kgm., given as a 10 per cent. suspension or in 10 per cent. aqueous polyvinyl alcohol, caused a loss of weight. During one experiment the hair on the feet, legs and abdomen fell out. The haemoglobin never fell below 70 per cent. (Sahli).

In dogs anaemia was produced, but it was transient. One dog weighing 10·5 lb. was given four daily doses of 5 gm. The haemoglobin was 130 per cent. before treatment and fell to 40 per cent. after it and then increased to 100 per cent. four weeks after the beginning of treatment; the dog was very lively throughout and gained 4·5 lb. in weight. Two other dogs weighing 15·5 lb. and 20 lb. were given a total of 48 gm. each in six daily doses of 5 gm. followed by four daily doses of 2 gm. Marked anaemia followed, with anisocytosis, poikilocytosis and numerous nucleated red cells. The haemoglobin of the dog weighing 15·5 lb. was 110 per cent. before treatment and it fell to 20 per cent. with 1·35 million red cells and 25 per cent. of reticulocytes at the height of the anaemia; this dog was very weak, but the symptoms disappeared when the dose was reduced to 1 gm. a day. After the total of 48 gm. had been given its haemoglobin had risen to 37 per cent. and the red cells to 3·13 millions. The haemoglobin of the dog weighing 20 lb. was 80 per cent. before treatment began and it fell to 35 per cent. with 2·06 millions of red cells. After the total of 48 gm. had been given the haemoglobin was 50 per cent. and the red cells 2·7 millions. After four days of rest both dogs were given an additional 4 gm. daily until 64 gm. had been given, *i.e.* each had then had 112 gm. But anaemia could not again be produced by this second course of the drug, during which the lowest haemoglobin value of the dog weighing 15·5 lb. was 37 per cent. and lowest haemoglobin value of the dog weighing 20 lb. 53 per cent. Six days after the last dose these haemoglobin values were 55 and 75 per cent. respectively. At the end of the experiment the weights of the dogs were practically the same as at the beginning.

The low toxicity of phenothiazine is due to its rapid elimination. Reviewing the literature the authors refer to the statement of SWALES and COLLIER that 50 per cent. of the phenothiazine given is excreted unchanged in the faeces. Most of the remainder is excreted in the urine in an oxidized form which is also found in the blood, the concentration curve in the blood being almost parallel to that in the urine. The authors determined the total amount of phenothiazine derivatives in the urine by diluting 1 cc. of urine with 7 cc. of water, adding 1·5 cc. of conc. HCl and 0·5 cc. of 30 per cent. hydrogen peroxide; after standing for 20 minutes the thionol formed in this mixture is estimated by comparing its colour with that of solutions of thionol of known strength. They found that a single dose of 1·5 gm. of phenothiazine is almost completely eliminated in 54 hours, most of it being eliminated

in the first 8 hours, with a second peak at 25–30 hours ; after 4–5 days no more of the drug could be detected. After 5·5 gm., given over 4 days, elimination was complete 6 days after the last dose, the bulk being excreted before the fourth day. *G. Lapage.*

HUMPHREYS (D. R.). **Death from Phenothiazine Poisoning.**—*Lancet.* 1942. July 11. pp. 39–40.

This is apparently an account of the same case as that reported in the *Lancet*, 1942, Jan. 17, p. 86. [See this *Bulletin*, 1942, Vol. 39, p. 193.] *C. W.*

ESKOLA (O.). Ueber die Giftigkeit der Wurmmittel im Lichte von Bilirubinuntersuchungen. [**On the Toxicity of Worm Remedies in the Light of Bilirubin Investigations.**]—*Acta Med. Scandinavica.* 1942. Vol. 109. Nos. 5–6. pp. 548–565. With 6 figs.

Eskola treated 40 people in whose faeces worm eggs (presumably cestode eggs) had been found or who had been treated for worms (unspecified) with Filix mas, Gland. kamala, Semina cucurbitae or Extr. aspidii spinulosi, with castor oil or magnesium sulphate as aperients ; a few said that they had had worms, although no worm eggs could be found in their faeces. All were free from other disease, special care having been taken to exclude affections of the liver and the blood.

Eskola believes, in spite of the comparative scarcity of his material, that 3·5 gm. of Filix mas or of Extr. aspidii spinulosi, 10 gm. of Gland. kamala or 150–200 Semina cucurbitae suffice for the expulsion of worms (presumably the author means tapeworms only). No bilirubin or urobilin was found in the urine of any of the patients and urobilinogen was present only in slight amount in a few of the urine tests. Skin icterus was present in only one patient. Most worm remedies increase the colour and bilirubin content of the serum temporarily and parallel with one another. The diazo reaction remains negative ; the toxic action of the worm remedies on the liver cells hinders the excretion of blood pigment. The composition of male fern and its toxicity are discussed. To the host, Extr. filicis is the most toxic of the remedies used, Extr. aspidii spinulosi is less toxic and Gland. kamala only slightly so ; Semina cucurbitae are relatively non-toxic. The toxicity is rather greater if castor oil is used as an aperient than if magnesium sulphate is used. While 3·5 gm. of Extr. filicis or Extr. aspidii spinulosi seem only slightly toxic to patients, the addition of only 0·5 g. of Filix mas to the dose increases its toxicity relatively strongly. GRAWITZ, on the other hand, gives 8–10 gm., of Filix mas as the suitable dose and CLOETTA states that more than 8 gm. should not be given. The use of 3·5–4 gm. in Finland is explained by the higher content of the active principle in the Finnish male fern.

Prolonged administration of atophan to 4 patients in daily doses of 1·5–3·0 gm. did not cause any increase in the pigment and bilirubin content of the blood ; other authors quoted by Eskola have stated that intravenous injection of atophan increases the bile pigment in the gall bladder by its direct action on the liver cells. *G. Lapage.*

Scott (J. Allen). **The Epidemiology of Schistosomiasis in Venezuela.** —*Amer. Jl. Hyg.* 1942. May. Vol. 35. No. 3. pp. 337–366. With 2 figs. [25 refs.]

This is one of a series of papers recording the results of the author's investigation, done at the request of the Venezuelan Government, of schistosomiasis (*S. mansoni*) in Venezuela. After working for three years the author concludes that schistosomiasis is one of the most important public health problems there. It is at present impossible to assess its seriousness accurately, but a rough estimate puts the number of infested persons at 30,000. In the different communities examined various grades of infestation were found: in some places about 25 per cent. of the population were infested, in other areas all the people had become infested by the age of 5–10 years.

Detailed data about the geographical distribution of the disease are still being collected, but enough information has been obtained to show that schistosomiasis is restricted to one endemic area only, namely, the central part of the northern coastal range. The reason for this is apparently the limited distribution of the intermediate host, the planorbid snail *Australorbis glabratus* (*Planorbis guadeloupensis*). No new foci of first-class importance are likely to develop unless the snails become more widely distributed and the practice of irrigation extends.

The conditions favourable to the snail can usually be recognized by an experienced observer. The snail must have either one long period of water in ditches, irrigation canals, etc., to enable a new generation to grow up sufficiently to withstand natural drying up of its environment, or the young snails must have protection against the dry periods which occur when irrigation canals are not used every day. They withstand slow, natural drying quite well and survive in canals used only a few days each week, but do not survive in appreciable numbers when the canals dry up quickly and completely between irrigation periods. Most snails are found in stagnant water, in residual pools left after the use of irrigation canals and in slow-moving water. If the local soil allows the growth of large aquatic plants, these help the snails by reducing the speed of the water currents and by providing food for them. Where the large plants are cleared, the snails may still be abundant, feeding on algae on the sides and bottoms of the canals.

Infestation of human beings seems to be at least as severe as the author found it to be in the peasants of Egypt. Infestation is commonest where sugar cane is irrigated in the broad portions of the mountain valleys. Wherever in this region the occurrence of sandy, well-drained soil does not favour the snails, the infestation does not exist. Although the snails occur in Lake Valencia, few people make contact with the water of this lake and, where they do, the vegetation has been cleared from the shores and no snails are found there.

Elsewhere, in every rural community where snails exist, at least a quarter of the population are infested. In most places nearly all the men and three-quarters of the women are infested, because the men have prolonged and more frequent contact with snail-infested water in the fields. Pollution of this field-water by defaecation is widespread; near the houses deposited faeces are washed by the rain into the canals. It therefore happens that, wherever snails occur near houses, virtually all the women are as highly infested as the men and the children acquire early infestation. Such communities harbour large numbers of worms. Persons infested early in life go on passing eggs for the rest of their lives

The percentage of infested snails is a measure of the degree of pollution during the preceding month ; more than 5–10 per cent. of the snails are seldom infested at any one time and place, although occasionally 25 per cent. were found infested ; and in one densely populated suburb 70 per cent. of the snails were always found to be infested. Negative examinations of snails are of little value.

Egyptian experience has shown that large-scale treatment of the infested people is not worth while. Other measures are difficult and expensive. It is, for example, easy to make latrines, but difficult to enforce the use of them. It is even more difficult to devise sanitation that will not pollute the irrigation water. The only hopeful control measure is the reduction of the number of snails. The ecological study of the shail made by the author indicates that minor changes in irrigation practice might effect this and such changes would also have agricultural value. These changes are discussed by the author in an earlier paper [this *Bulletin*, 1940, Vol. 37, p. 484]. A larger plan of control on these lines is being prepared. *G. Lapage.*

Brumpt (E.). Observations biologiques diverses concernant *Planorbis* (*Australorbis*) *glabratus*, hôte intermédiaire de *Schistosoma mansoni*. [**Various Biological Observations concerning** *Planorbis* (*Australorbis*) *glabratus*, **Intermediate Host of** *Schistosoma mansoni*.]—*Ann. Parasit. Humaine et Comparée.* 1941. Vol. 18. Nos. 1–2–3. pp. 9–44. With 7 figs. & 1 plate. [50 refs.]

Prophylaxis against intestinal bilharzia due to *S. mansoni* would be easy if it were possible to prevent the contamination with human faeces of water in which the snail intermediate hosts live ; but it is impossible to prevent agricultural workers from defaecating in plantations from which tropical showers wash faeces into the irrigation canals where the planorbids abound. Treatment of the patients has given only mediocre results, so that an attack on the snails should be made. For this an exact knowledge of the biology of the snails is necessary and this paper records work done by Brumpt on the habitat, resistance and reproduction of *Planorbis glabratus*.

Pilsbry established the identity of *P. glabratus* and *guadeloupensis* and the name *guadeloupensis*, so well known, must unfortunately disappear according to the rules of zoological nomenclature.

Habitat.—The snail is found by preference in natural or artificial fresh water canals in which the water moves slowly and is often fouled by human faeces. Hundreds may be found quickly in canals on clay soil in which the water is stagnant, while in cemented canals, such as those seen in the Caracas region of Venezuela, in which the water flows quickly, only isolated individuals are found on the walls. Like most planorbids, *P. glabratus* comes up to the surface when the plants or other supports on which it lives are shaken. When laboratory cultures kept at 25°C. are shaken only about 30 per cent. of the snails fall to the bottom by expelling air from their pulmonary cavities, but at 32° or 37°C. almost all of them remain at the surface when the cultures are shaken.

Resistance.—Unlike other species of Planorbis, *P. glabratus* can live in brackish water. Brumpt found that it could not live in cultures in Paris containing more than 25 per cent. of artificial sea water ; but, if snails kept in brackish water till their bodies bulge out of their shells and they seem to be about to die are put back into fresh water, they

become normal and lay eggs two weeks later. In water containing 20 per cent. of artificial sea water they do not feed or lay eggs, but they can live for about 35 days. When eggs laid in fresh water are put into water containing 25 per cent. of sea water they do not develop.

In nature *P. glabratus* withstands a day temperature which is often above 37°C. ; but, during the night, or in pools that are shaded for part of the day, the temperature falls to about 25°C. and the snails can recover from effects of heat. In the laboratory a constant temperature of 37°C. kills the snails in 3 to 4 days, even if they have been gradually accustomed to it. From a continuous temperature of 32 33°C. they still suffer, eating little and not laying eggs ; but a temperature of 33° or 37°C. for only a part of the day does not prevent them from living and laying eggs normally. When they emit cercariae they are much more sensitive to the action of solar heat. They rarely have to experience cold in nature and do not resist it well. Specimens of all ages taken gradually down to 4 to 5°C. died in four to five days. At 10°C. they live but do not feed and group themselves in voluminous masses. At 15°C. they feed much less than at higher temperatures. The optimum temperature for cultures is 25–30°C.

In nature they often withstand the drying up of the drainage canals in the plantations. Out of 192 snails taken from a canal at Villa de Cura in Venezuela which had been completely dried for more than a week [a photograph of this canal is given] 25 per cent. revived. But of 95 specimens rather smaller in size taken on November 6th in the dried clay of Caracas and put in water in Paris on December 4th (28 days later) 30 were still alive and laid eggs a few days later, but none emitted cercariae of *S. mansoni*, a fact which suggests that infested planorbids resist drying less efficiently ; the dried planorbids became active one or two hours, or rarely 24 hours, after they were put in water at 28–30°C. In the laboratory 21 out of 44 snails resisted 50 days of drying more severe than is encountered in nature, where the humidity of the subsoil, accidental rain, etc., may prevent complete desiccation. Probably in nature they can live much longer under dry conditions when they are sheltered by vegetation ; in this respect they resemble *P. boissyi* in Egypt, which can, according to BARLOW, resist drying in nature for at least two months and for about six months in the laboratory in metal boxes with a little dry earth. When they are dried up the snails inhabit the bottom of the shell, where the body is reduced to a sixth or a seventh of its normal volume.

Planorbids parasitized with cercariae are less resistant to drying. Barlow showed that this was true of *P. boissyi* in Egypt infested with *S. mansoni* and CORT demonstrated it for *Oncomelania nosophora* infested with *S. japonicum*. Brumpt showed that it is also true of *P. glabratus* and thinks it certain that the larval schistosomes damage the snails. Out of 150 specimens collected at Guadeloupe on July 22nd, 1939, of which 30 per cent. were infested when they were examined in Colombia on August 14th, only 30 survived and these were not infested.

Erosion of the shells, in the form of circular or linear erosions of a pearly colour on the olive green or brown shell, were always present, being most numerous on the older coils at the centre of the shell, where actual perforations may occur. These were seen in cultures in the laboratory as well as in snails collected in the field, but in cultures they were present only when several snails were cultured together. Brumpt has found similar erosions on the shells of *Planorbis exustus*, individuals of which species browse on each other's shells and damage

them ; in these experiments with *P. exustus* corrosive action of the water was eliminated and Brumpt seems to think that the most likely explanation is that algae and related organisms extend the damage done by the snails themselves to each other's shells.

Reproduction.—Copulation is easily observed, the snails uniting unilaterally in chains, the middle snail of three acting as female to the first one and as male to the one behind it ; the process may continue for a long time, but towards the third day sexual excitement seems to die away and after that only rare brief pairing occurs. Discussing the question of self-fertilization in pulmonates, Brumpt gives his own illustrations of the eggs of *Bulinus contortus* showing eggs with two polar bodies, the presence of which necessitates the view that they must be fertilized by a sperm. He gives a table of all the Basommatophora in which self-fertilization has been proved ; this includes *Bulinus contortus, Planorbis glabratus* and *P. boissyi*. The fecundity of different individuals of *P. glabratus* of the same size and kept under the same conditions varied greatly, but snails 65 days old at the end of one experiment laid an average of 23·3 eggs per egg mass and 179 snails laid 4,171 eggs. The rhythm of egg laying was not regular ; some laid almost every day for several days, stopping for two or three days and beginning again ; others laid two or three egg masses a day and then stopped for several days. Some egg masses that were apparently abnormal did not develop and some eggs in fertile egg masses did not develop. The egg mass is like that of other planorbids, flat and oval with a hilus indicating the beginning and end of the whitish egg ribbon. The eggs are in one plane, compressed against each other in rows of one, two, three or four, according to the age of the snails, and are covered by a resistant elastic capsule with a horny colour. The number of eggs varies with the size of the snail. A snail of medium size (24 mm.) lays first masses which hold 5–20 eggs. Normally egg masses are laid quickly (in 3–5 minutes) and at night. The snails will not lay in water containing gases of putrefaction, but they lay in less than an hour after they are put into fresh water. In nature the egg masses are generally laid on plants and various solid bodies and on the shells of other planorbids. In cultures they will be laid on Cellophane or cover slips and can then be examined on these, or on the walls of the glass container, when 90 per cent. of them are laid at the level of the water or within its upper third. The optimum temperature for egg laying is 20–25°C. ; it ceases at temperatures above 30°C. and below 15°C. Lowering the temperature stimulates the deposition of eggs. The volume of water present has no influence on egg laying ; the effect of brackish water has already been noted. When individuals are isolated they lay rather more egg masses. Infestation with larval stages of *S. mansoni* effectively reduces the egg-laying capacity ; during the first few days of parasitism they lay as many eggs as the uninfested controls ; towards the tenth day, even when tentacular lesions exist, the egg-laying continues ; but towards the 18th day, at the moment when the daughter sporocysts emigrate, egg-laying practically ceases and occurs only rarely during the emission of cercariae. Complete castration due to the presence of the parasites was never seen in spite of the abundance of sporocysts and cercariae in the genital glands. Eggs become rarer and rarer, but spermatogenesis continues and sperms are numerous. Cercariae may occur inside the eggs and figures of them in this situation are given. Inside the eggs they live for a maximum of 24–36 hours and their dead bodies are eaten by the young snails when these develop. The presence

of living or dead cercariae in the eggs does not as a rule seem to damage the development of the young snails, although the active movements of cercariae may damage the segmenting eggs and this may perhaps produce monstrosities. Their presence in the eggs shows that they use the female genital ducts as a means of getting out, as well as other routes. A heavily infested egg mass was found laid in water which did not contain any cercariae, so that the cercariae found in eggs had not entered them after they had been laid: further, when cercariae penetrate anything, they drop their tail appendage and this is always present on those found inside eggs; when millions of cercariae were given the opportunity to penetrate egg masses they showed no attraction to these and did not try to penetrate them.

P. glabratus, when they have, at the age of 6 weeks, reached only half their average diameter (10–11 mm.; mature individuals reach 24–26 mm.) may lay eggs and they seem to go on doing so until they die. At 25°C. in cultures they can lay during an average of 15 months about 20 egg masses of 50–80 eggs per month; this amounts to about 20,000 eggs during their lifetime. But natural enemies in nature control this multiplication. When the water is often renewed to prevent the depredations of infusoria, rotifers, oligochaetes and various crustacea, 85 or even 100 per cent. of the eggs may hatch. Resistance of the eggs to drying varies according to the stage of evolution of the egg. Eggs laid outside the water in a tube died in 2–3 days; eggs laid in fresh water and taken gradually into water containing 25 per cent. of sea water did not develop; eggs in a humid atmosphere in a tube kept at 25°C. continued part of their development and the embryos emerged when water was added, but young eggs did not develop. Snails isolated from birth developed much quicker than those that were kept in communities. The extent of the water surface does not seem to affect the rate of growth, because snails isolated in tubes are always bigger than others kept in communities in larger vessels. Reproduction occurred at all times of the year at 25°C. and 6 generations a year are possible.

In Venezuela intestinal bilharzia is localized and only some 20,000–25,000 persons are infested; but in Brazil, Guiana and the Antilles several millions are probably infested. Natural infestation of *P. glabratus* is sometimes intense; it may reach the degree of experimental infestation, which is often 100 per cent.; but sources of infestation vary so much that areas are often found where no snails are infested and others where the infestation is high. Brumpt published in 1940 his work on the evolution of the parasite in the snail, which completed the work of Leiper, Lutz, Lampe and Faust and Hoffmann. Here he gives figures of the presence of cercariae in the tentacles, which show that emigration of the daughter sporocysts into the hepato-pancreas, although normal, is not necessary.

A section on methods to be used for the destruction of the snails completes the paper. These include the removal of useless collections of water, regular pumping, drainage, control of irrigation canals and the covering of canals and drains where possible. Sea water could be used, but if it is to be successful it must act for several successive days at a temperature of 35–40°C. Copper sulphate, carbonate of copper, quicklime and ammonium sulphate could be used, because all molluscs are susceptible to the action of these. Among biological methods alternate drying and filling of the canals every 15 days is not effective, as the above study of the resistance of the snails to drying shows. It is

better to help the multiplication of their natural enemies. These are considered in turn, but among them ducks appear to be the only ones likely to be of practical value. No parasitic trematode is known that can castrate the snails completely. *G. Lapage.*

ZELLWEGER (Hans). Ueber die durch Schistosomumintercalatum hervorgerufene Intestinal-Bilharziose im Gabun. [**Intestinal Bilharzia in Gabon due to** *Schistosoma intercalatum*.]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1940. Nov. Vol. 44. No. 11. pp. 507–520. With 3 figs. & 1 map. [12 refs.]

Intestinal bilharzia caused by *Schistosoma intercalatum* is more frequent in Gabon than it was formerly thought to be. Its distribution is bound up with the course of the River Ogowé and its communicating tributaries and lakes. A description of these is given, with a map illustrating the area investigated by the author from his base at Lambaréné. The heaviest infestation occurs on the River Ogowé between N'Djolé and Achouka about 100 km. above the entry of the Ogowé into the Atlantic, including the tributaries and communicating lakes in this area ; no certain cases were found above N'Djolé. The question whether the infested area in Gabon is connected with the area of the Belgian Congo also infested with *S. intercalatum* is discussed. CHOUBERT thought that all the geological and petrological evidence suggested that the river Congo once flowed through the bed of the Ogowé. Zellweger thinks that the more rapid current in the river Ogowé in its upper half above N'Djolé may prevent the intermediate host from breeding in these upper waters and may confine it to the slower waters of the infested area defined above.

Hospital work left no time for a study of the intermediate host, which is in the Belgian Congo, according to VAN DEN BERGHE, *Physopsis globosa*. Zellweger found *Physopsis africana* in Lake Zilé, one of the lakes in the most heavily infested area.

Tables are given showing the frequency of the occurrence of the parasite. In the hospital 153 cases were found among 21,128 patients between 1931 and 1937, while between October 1937 and April 1939 there were 274 cases among 6,560 patients. This relative and absolute increase was thought to be due partly to a more intensive search for the eggs and partly also to an increase during these months in the number of cases. Examination of the faeces of school-children showed that 33·8 per cent. harboured schistosomes and 19·2 per cent. had no parasites at all. There was an enormous infestation with intestinal parasites, but rare complaints of abdominal trouble. Zellweger's studies convinced him that the threshold of stimuli exciting complaints of such troubles is much higher among the natives than among the whites and also that the natives forget severe attacks of pain in a few days. Chronic and subchronic bilharzia can exist for long periods without pain. In the Belgian Congo van den Berghe found 50 per cent. of the school-children of the Yakusu Mission near Stanleyville infested with *S. intercalatum*.

Studying race influence Zellweger found that most of the carriers of the parasite belonged to the Pahouins or Fangs (*e.g.*, 276 out of 427 carriers were Fangs), who are the largest tribe in the Ogowé area and are more often fishermen than other tribes are. Of the school-children examined 189 were Pahouins and 40·7 per cent. of these were carriers while 20·4 per cent. of children of other tribes were carriers. NESSMANN

and TRENSZ found that most of the patients having this parasite were Pahouins.

The symptoms are described, but the author remarks that it is very often difficult to be sure that any particular symptom belongs to this disease. Almost all the natives have intestinal parasites and malaria and often suffer from syphilis, gonorrhoea and *Loa loa*. Heavy infestations with *S. intercalatum* begin with high fever between 39° and 41°C., marked malaise, pains in the upper abdomen, enlarged spleen and liver and more or less icterus. Bronchitic symptoms with a leucocytosis and slight eosinophilia, the cough disappearing after treatment with fouadin, are described in two cases. The liver and intestinal symptoms have a similar course and prognosis to those caused by *S. mansoni*. Allergic symptoms, such as urticaria and bronchial asthma, are described, but the author could not be sure that these were causally related to the bilharzia.

Subacute conditions may follow the acute stages, when formed stools gradually appear containing layers of blood and mucus; there may then be severe abdominal pain and tenderness, especially in the lower abdomen and along the colon. One 6-year-old girl had typical symptoms of appendicitis which disappeared after two injections of antimony. After some weeks or months the pain and blood and mucus may disappear, but a self cure, such as has been described in cases of infestations with *S. mansoni* and *S. haematobium*, is hard to prove. Eggs are often found only after several stool examinations by concentration methods; fouadin can be given to provoke their extrusion.

In the chronic stage colitis with watery, not bloody, mucus was found in five women, all of whom were very emaciated; after prolonged search eggs were found, some of which were dead. Three were cured with 70 cc. of fouadin combined with rivanolein; one was improved but not cured; one died and autopsy revealed a rectal stricture which would not allow the passage of the little finger, and eggs in the urine and stool, with peritonitis, colitis and adhesions between the large omentum and parietal peritoneum; there was also cystitis and slight bilateral hydronephrosis.

The bladder is, however, seldom affected, though rectal polyps may occur. Dysmenorrhoea occurs fairly often, but this may be due to the almost ubiquitous gonorrhoea and its complications, but there remains a great preponderance of dysmenorrhoea without gynaecological troubles. Exacerbations of abdominal pain often occur during pregnancy, beginning mostly in the second month with blood and mucus in the stool; in these cases there is a tendency to miscarriage in the second half of pregnancy.

For treatment fouadin, anthiomaline and a preparation called "Versuchspreparat 1174/I + II" and tartar emetic were used. These were equally good, but fouadin and anthiomaline were rather less toxic than the other preparation; tartar emetic was soon given up. The usual doses were given, but two or three treatments were needed and the author is very doubtful whether a cure is effected. Parasites in the liver are not affected by therapy and relapses may be explained by this fact.

S. mansoni was found in two women and one child; the women had come to Gabon from Dahomey and the Cameroons respectively, and the child had come from the French Congo.

S. haematobium was found in one man, who had come from Togo.

G. Lapage.

CHOUDHURY (L. M.) & LAHA (P. N.). **A Hydatid Cyst of the Spleen.**—*Indian Med. Gaz.* 1942. Jan. Vol. 77. No. 1. pp. 29–30.

LAZAREVIĆ (Vojin). Mächtige Nierenechinokokkencyste mit völligem Schwund des Nierenparenchyms. [**Large Hydatid of the Kidney.**]—*Deut. Ztschr. f. Chirurgie.* 1942. July 20. Vol. 255. No. 11 & 12. pp. 747–750.

CANNON (D. A.). **A Case of Human Infection with a Species of** *Coenurus.*—*Ann. Trop. Med. & Parasit.* 1942. June 30. Vol. 36. Nos. 1 & 2. pp. 32–34. With 5 figs. on 2 plates.

An African male, aged 30, complained of stiffness and discomfort in the right forearm when he used his rifle. A smooth, elastic, rounded, rather tender swelling, one inch in diameter, was found; puncture failed to withdraw fluid. Under 2 per cent. novocaine anaesthesia a colourless jelly-like mass was excised, which proved, when sectioned, to be a coenurus cyst, the species of which could not be determined. A slender fibroareolar capsule enclosed a very irregular space into which projected numerous scolices, each of which had four suckers, a rostellum and double row of sickle-shaped hooks, only one of which was completely preserved (its length was about 137 microns). References are given to six other cases of coenurus cysts found in man; at least one of these occurred in the brain and caused Jacksonian epilepsy and other symptoms. *G. Lapage.*

WILSON (Paul W.). **Maturation of Ascaris Ova in Sea Water; a Possible Factor in Dissemination of Ascariasis in American Samoa.**—*Amer. Jl. Trop. Med.* 1942. May. Vol. 22. No. 3. pp. 305–307.

Stool surveys of 996 native Samoans belonging to widely scattered groups showed an incidence of 86·4 per cent. of *Ascaris lumbricoides*, 71·0 per cent. of *Trichuris trichiura*, 18·1 per cent. of *Necator americanus*, 0·5 per cent. of *Strongyloides stercoralis*, and 0·13 per cent. of *Enterobius vermicularis*. Excepting about 400 people in inland villages all the 13,000 Samoans live in coastal villages. In several coastal villages where the latrine facilities had been considered adequate there was 100 per cent. incidence of ascariasis.

Natives of the coast of American Samoa universally season their food with sea water instead of with salt; the food is usually "dunked" in fresh sea water just before it is eaten and always after it has been cooked. After a hard day's work or any exertion causing loss of salt by perspiration the natives also drink a mixture of equal parts of coconut milk and sea water taken freshly from the sea a few paces from the waterline in front of the village. There is also the further age-old custom of defaecating in the sea on the beaches, which usually consist of flat coral shelves covered at high tide with only a foot or more of water which is relatively quiet, so that removal of débris from it is sluggish. The eggs of both Ascaris and Trichuris are heavier than sea water, which, at Samoa, has a specific gravity of 1·026, so that it might be more than 10 days before these eggs would find their way to deep water beyond the coral shelves; the roughness of the coral would itself hinder their removal. The author found experimentally that eggs of Ascaris would develop until the active larva was formed in 10 days in sea water aerated daily and containing 2 per cent. formalin at a room temperature of 80–88°F. Controls in fresh water took

the same time to develop to the same stage. "Live" eggs of Ascaris and Trichuris were found in shallow pools in the coral at low tide, 60–75 feet from a beach latrine. Sea water taken from coral beach pools at low tide, at a point about 20 feet to one side of the outlet of the discharge pipe from latrines, contained 20 "live" eggs of Ascaris in various stages of development and one egg of Trichuris per gallon of sea water.

In four villages, by the Willis brine floatation technique, 100 per cent. incidence of Ascaris was found; in another, which had no flat coral beach, but deep water even at low tide, the incidence was 65·3 per cent.; in another, which lay about three miles inland on the Bay of Pago Pago, the cookhouses had enough salt to season their food and sea water was not used, because the villagers did not like the taste given by the sea water, which they attributed to faecal contamination, and the incidence here was 66·5 per cent.

The author thinks his results justify the assumption that Ascaris infestation from the sea water may account for some of the infestation and reinfestation of natives in Samoa. *G. Lapage.*

Junge (Werner). Die operative Behandlung der Elephantiasis des Beines. [**The Operative Treatment of Elephantiasis of the Leg.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1940. Dec. Vol. 44. No. 12. pp. 549–562. With 6 figs.

In spite of all the improvements in internal therapy the treatment of elephantiasis is still purely surgical. Surveying existing operative technique, the author agrees with Vogel's view that operation on the scrotum gives excellent results, but the results of operation on the extremities are rarely satisfying. Amputation has been rightly condemned. Plastic restoration of the limb has been sought by reduction of its circumference and by the provision of new lymph channels for the promotion of a better flow of lymph. The various methods of reducing the circumference of the limb are briefly described. Manson's method of making strips of skin 5–10 cm. broad and 20 cm. long and then removing the subcutaneous elephantiasis tissue is not satisfactory because such strips have too narrow a stalk and become necrotic, being badly nourished. It is also wrong to open the marrow cavity of the bones in order to produce a new flow of lymph; this procedure exposes the marrow to risks of infection from the elephantiasis tissue, which is never sterile. The author has never found that the musculature is affected. Knott and Rice have shown that it is wrong to assume that the operation should be so radical that only healthy tissue remains. Another failing of existing methods is the insufficiency of the precautions taken. The principle of the operation must be the most thorough removal possible of all inflamed proliferative tissue and the creation of new lymph channels.

Junge has developed a new technique and has used it, with slight changes, for 6 years. Out of 26 cases which he was able to follow throughout most of this time, only three relapsed and the relapse of one of these was so slight that another operation was not advisable. About 30 other cases passed out of his control and could not be followed up. The cosmetic and functional results were good throughout; in almost all cases the result was a limb very similar to the healthy one.

Junge's technique varies according to the part of the leg affected and the form taken by the disease. There are at least three methods corresponding to the division of the limb into thigh, leg and foot; and the disease can be divided into the cylindrical form, which may be oedematous or fibrous, and the irregular form, which may be sulcate (furrowed), lobated or tuberous.

The lower leg is most often affected and the commonest form here is the cylindrical form. Newly developed cases are mostly oedematous and older cases are fibrous and more pronounced. In oedematous cylindrical cases he found it wise not to resort to the knife too early or without due consideration. Conservative methods were best for these cases. Baths, massage, bandaging and elastic stockings gave, as MANSON showed, good results. When such methods fail operation may be considered, especially if the patient's capacity for work is seriously endangered. The main object of operation on oedematous cases is the creation of new lymph channels. Conservative therapy is, on the other hand, rarely needed for the fibrous cylindrical form. Operation is made necessary by the persistent relapsing lymphangitis and its attendant fever, together with the weight and deformity of the limb and the reduction of the capacity for work. The object of the operation is not so much to repair lymph channels as to remove the elephantiasis tissue.

The author's method for operation on the lower leg, whether the disease takes the oedematous or the fibrous cylindrical form, is as follows :—

Before the operation all ulcers, pustules, open skin wounds, etc., must be healed up and the skin got into a healthy state. Baths, massage and vigorous rubbing of the skin with alcohol stimulate the blood supply to the skin. By night the limb is raised and by day bandaged. For operation the patient lies on his back with the leg as for amputation; the skin is painted with alcohol, iodine being avoided in order to preserve the skin, which is important for the success of the operation.

A slightly curved incision is made from the head of the tibia to the ankle joint; two further incisions at the upper and lower ends of this make y-shaped incisions at these points. The skin is now separated on both sides laterally as far as possible, extension of the initial incisions being made if necessary. One-third of the skin of the whole leg circumference must be left firmly adherent to the calf. Both flaps of skin are then drawn to the sides. The elephantiasis tissue thus exposed is now incised along the tibial margin as far as the periosteum and a similar incision is made, parallel to this and at a distance from it, down to the muscle fasciae; these two incisions are joined by a transverse incision at their upper ends. The piece of tissue thus marked out is hooked up at its upper border, pulled away and separated from the underlying muscle fasciae. This separation is repeated in ring-like strips down the leg until none of the proliferated tissue remains except the part of it behind on the calf which is still connected with unseparated skin. During this part of the operation a great deal of dammed-up lymph flows away. The proliferated tissue left on the calf shrinks. The limb is thus reduced to its normal size and, if the subsequent operation designed to establish new lymph channels is well done, there is no subsequent oedematous seeping causing renewed swelling.

To restore the flow of lymph about 25 windows are cut in the muscle fasciae over the whole of the leg, each rather bigger than a postage stamp, but none is cut over the tibia. Into each of these

windows a double silk thread is put by passing the needle deep through the muscles. The hanging ends of these are arranged like candle wicks to suck out lymph over the widest possible area of the leg.

The skin flaps are then replaced. If they are now too long, they are shortened ; and this affords an opportunity to remove any diseased skin. It is better to shorten both flaps than to leave only one, because this may necrose. Exact suture and insertion of an iodized gauze drain in the angle of the upper y-shaped incision completes the operation. Afterwards the limb should be splinted and kept raised and firmly bandaged to avoid secondary haemorrhage. After 48 hours the bandages are released only enough to enable the gauze drain to be removed ; the first change of bandages is after 6–8 days.

Complications were rare ; serious secondary haemorrhage practically never occurred ; but punctilious asepsis is very important. The two skin flaps, having broader bases than those used by Manson, are better nourished and only rarely necrose. They may have to be 15 cm. in diameter and the beginner often shrinks from making them ; but only in a few of the author's cases did necrosis of strips of skin 1–2 cm. broad along the tibial edge occur and this was harmless and quickly disappeared with treatment with zinc ointment. A scar forms over these areas, and the defect can be made good with Thiersch grafts.

Operation on the irregular form of elephantiasis of the leg is not so simple. The sulcate form may present, in the region of the foot joints, a number of deep invaginations of the skin going down almost to the muscle fasciae, or the leg may be encircled by ring-like invaginations which do not go so deep. If the grooves are not deeper than about 0·5 cm. they can be neglected, the operation being like that described for the cylindrical form. Deeper sulci are excised by incisions parallel to them and then the operation proceeds as before. If several sulci exist near together it is better to excise them all, and skin grafts may then be necessary. The lobate form may hamper the technique even more and the curved incision described above may not be possible. Lemoine was the first to use the wedge-shaped excision of stalked or lobated tumours of this kind at several sittings, but Junge thinks it better, if possible, to excise them at one sitting, in order to avoid the risks of lymphangitis. The incisions must depend on the position and size of the lobes and swellings and the only directions that can be given are that the principles must be to reduce the size of the limb and to promote new lymph channels, to aim at altering the complicated form into a simple one, to choose incisions that give skin flaps with broad bases and to avoid the use of the skin on the lobes for mending defects, because it may be diseased.

Operation on the tuberous form is almost impossible. In this form the pathological process is mostly in the skin itself, which is very much thickened to form tuberosities, rolls and nodules. It is practically impossible to save skin to repair defects at the end of the operation. In such cases the author tried Connor's method with quite good results. This method removes all the thickened tissue and covers the defective area with Thiersch grafts.

The method used for the leg cannot be used for the foot, because the anatomy is so different. In the foot the cylindrical and irregular forms occur, although, as the foot is usually uniformly affected and as its shape is different, the term cylindrical may be replaced by the term uniform. The operation described above for the oedematous form does not give good results in the foot, because the silk threads inserted

to promote the flow of lymph work well only near the foot joints ; they do not work well in the tendinous parts and all Junge's attempts to drain the dorsum of the foot failed. These cases were always treated by conservative methods. Once the swelling can be reduced sufficiently to permit the wearing of boots, much is accomplished and boots should be worn to prevent the return of the swelling. It is much more difficult to decide whether to operate on the longer-standing fibrous form, and the author thinks that operation should only be done on patients with this form whose capacity for work is very seriously limited or is abolished. Renewal of the lymph flow is not possible, and in all his cases the removal of the affected tissue without lymph drainage resulted in relapses, so that this method must be rejected. Removal of the skin and subcutaneous tissue with grafts to cover the skin deficiency give more permanent results.

For this operation special care of the skin is not necessary, nor is it necessary to reduce the volume of the foot by rest in bed and raising of the limb ; the whole foot may be painted with iodine. At the upper border of the proliferated tissue at about the level of the malleoli a circular incision is made round the ankle joint down to the fasciae. From this an incision is made in the middle line of the dorsum of the foot to the toes and a corresponding one from the Tendo Achillis to the lower border of the heel. All the diseased tissue and skin is separated from the fasciae on both sides of the incision up to the border of the foot. A horizontal incision along the lateral border of the foot separates all this, so that the flap hangs only from the toes ; if the toes are involved, the tissue of their upper sides is removed by traction on the flaps ; otherwise a transverse incision along the base of the toes separates the flaps. The foot is then covered with healthy skin from the thigh ; especially good covering of the ankle is necessary to prevent cicatrization here. Few difficulties occur with native patients who have such good powers of regeneration, but the author does not know whether this would apply to Europeans.

The irregular form is more frequent in the foot, being almost the rule. Sulcate, lobated and tuberous forms may occur together and the radical operation is then necessary. The tuberous form showed most relapses, which often took the form of small nodular excrescences like dermatitis verrucosa. Conservative treatment was adopted for lobate forms that affected only parts of the foot.

In the thigh the disease is least common ; it never appears without similar disease of the leg or foot ; it is seldom so extensive that it needs a special operation. The oedematous cylindrical form is the commonest. Usually lymph from the leg has been blocked in the thigh and after operation on the leg the disease may disappear from the thigh with palliative treatment. The fibrous form may need operation. A curved incision is made along the outer side over the fascia lata and the flap of skin left connected with the subcutaneous tissue and muscle fascia corresponds with the course of the saphenous vein. This avoids so far as possible damage to the superficial vessels. It can be assumed that the lymph flow on the inner side of the thigh still functions, so that attention to the lymph flow can be concentrated on the outer side, the fascial windows being made especially big. The author never saw the irregular form with skin invaginations in the thigh, but other authors have written of the lobate form. KNOTT limits himself to simple extirpation and covering the deficiency with the healthy skin. The cylindrical form then remaining can be dealt with by the

operation described above. The tuberous form can only be dealt with by Connor's method, but, as the wounds are then so large that they cannot be covered by grafts, this form is inoperable.

Combined disease of the thigh, leg and foot is best treated by operation first on the leg and then on the foot. Palliative methods may be sufficient to reduce the foot. The thigh is always done last.

G. Lapage.

HILLMAN (Robert W.). **Oxyuriasis of the Appendix : a Clinical Study of 31 Cases.**—*Brooklyn Hosp. Jl.* 1941. Apr. Vol. 3. No. 2. pp. 83–93. [22 refs.]

The author discusses 31 cases in which *Enterobius vermicularis* was found in the appendix by either gross or microscopical examination. These represented 2·39 per cent. of all cases operated upon for appendicitis during four years.

The infestation was found in both sexes and at all ages but was commonest in girls of school age or at adolescence. All the patients, except one negro, were whites with an average age (excluding the two aged 63) of 13·1 years, the youngest being 5 years old, and only six being older than 20 years. Only eight were regarded as being acutely ill, but 19 were regarded as emergencies. The cases were commoner in September and October and 22 cases occurred during the last six months of the year.

The usual history is of recurrent attacks of appendicitis during 1–2 years with mild abdominal pain, nausea and often vomiting for several days before admission ; the temperature is normal ; the patient is not acutely ill, being in general not so ill as patients suffering from appendicitis due to other causes ; but there is moderate abdominal tenderness and a slightly increased total white cell count with a relative increase of polymorphonuclears. The clinical picture varies, however, so much that the disease cannot be differentiated from acute suppurative appendicitis. There are usually no inflammatory changes and there is no characteristic pathological picture. Possibly the symptoms are due to hyperperistalsis of an appendix which is trying to expel the worms.

When a girl of school age has intermittent abdominal pains for 1–2 years and the clinical picture of mild to moderate appendicitis, Enterobius should be suspected as the cause. Generalized infestation of the lower intestine may be responsible for some of the symptoms, the more severe attacks of abdominal pain and vomiting being due to infestation of the appendix. The literature recording the incidence of Enterobius in the intestine and in the appendix is discussed.

G. Lapage.

DEFICIENCY DISEASES.

CHEN (Ji). **Nutritional Edema in Children.**—*Amer. Jl. Dis. Children.* 1942. Mar. Vol. 63. No. 3. pp. 552–580. [Refs. in footnotes.]

After a preliminary review of nutritional oedema the author gives data derived from a study of child refugees from the Sino-Japanese war zone. Most of the children had associated disease and had been living on diets deficient in protein, particularly animal protein, fat,

calcium, phosphates, iron and vitamins, especially the B complex, C, A, and D. Of 903 children, 433 (48 per cent.) showed clinical manifestations of nutritional disease and 130 (14 per cent.) had oedema apparently directly due to nutritional deficiency. Nutritional oedema was less frequent in infants than in older children, perhaps owing to the fact that many of the infants were fed on mothers' milk. There was little difference between the sexes except below 2 years of age, when females suffered from oedema more than males, probably owing to the greater care given in China to male infants. In the oedematous children there was a higher incidence of beriberi, tuberculosis, noma and lobar pneumonia, than in the non-oedematous children, who suffered more from typhoid, bronchopneumonia and rickets. The presence of oedema did not alter the general mortality rate but appeared to parallel a higher mortality in the infants under 2 years of age. The main causes of death in those with nutritional oedema were bronchopneumonia (31 per cent.), bacillary dysentery (27 per cent.) and noma (20 per cent.).

Of special interest was the presence of oedema of the brain, which was found in 50 per cent. of the autopsies and was thought to be due to more than the general tissue oedema. Adrenal changes, especially cortical atrophy, were also common and may have played some part in the production of the tissue oedema. The main factor in the production of oedema seemed, however, to be the low osmotic pressure in the blood due to lowering of the serum albumin and lack of compensation by the other proteins of the blood, but other factors are involved, as there were many patients with very low serum albumin values and without globulin compensation, who did not have oedema. Nevertheless, determination of serum albumin is the most important test in the study of nutritional oedema, all the oedematous patients showing values below the lower level of normal (4·5 per cent.) while 77 per cent. were below the critical level of 3 per cent. The serum globulin values of most of the oedematous patients, and of all the non-oedematous patients, were either normal or higher than normal, and the values were proportionately higher in the non-oedematous group. *F. Murgatroyd.*

RAO (M. V. Radhakrishna). **Intestinal Changes in Monkeys fed on Poor Rice Diets.**—*Indian Jl. Med. Res.* 1942. Apr. Vol. 30. No. 2. pp. 273–284. With 20 figs. on 5 plates. [12 refs.]

The object of this investigation was to observe the effect on monkeys of long-continued feeding on poor rice diets resembling those consumed by man in India. The production of any specific type of deficiency disease was not aimed at, but the most important observation was the tendency of the deficiently-fed monkeys to develop diarrhoea associated with specific change in the small intestine.

Young monkeys (*Macacus sinicus*) 4–7 pounds in weight were divided into two groups. Those of one were fed on a well-balanced lacto-vegetarian diet, resembling that consumed by certain races in North India. The other on a " poor S. Indian diet," resembling that consumed by poor rice-eaters, and given in a cooked state.

The first diet was superior in nearly all important food factors ; the second deficient in vitamins A, B and C and in calcium. Altogether 84 animals were observed ; 23 on the first, 61 on the second diet.

No significant differences in nutrition were noted during the first few weeks, but later on the health and well-being of the latter deteriorated. These animals soon became listless, lost energy and interest; some died within 6–8 weeks, others survived 6–12 months.

The appetite was impaired and there were attacks of diarrhoea with 4–10 small, pale stools a day. In animals thus affected a supplement of milk, sprouted grain and one banana a day usually checked the diarrhoea.

It was further noted that if the animals, as many did, contracted dysentery (with blood and mucus) those of the first group recovered quickly, but in the second it passed into subacute or chronic stage.

Some animals of the group showed signs of deficiency—catarrhal ophthalmia, xerosis of the conjunctiva, oedema of the eyelids, pyorrhoea, bleeding, sometimes ulceration of the gums, spastic or flaccid paralyses, and chronic ulcers.

The hair became coarse, sparse and "staring"—some showed symmetrical denudation over hands and feet with rough and scaly skin, hyperpigmentation and branny desquamation, suggestive of pellagra. The tail also showed similar changes.

A few showed oedema of the lower part of the face or scrotum, some of the hands and feet. The addition of dietary supplements cleared this up. The terminal clinical picture was one of great emaciation, prostration and chronic diarrhoea.

None of these changes was noted in the first group.

Pathological studies pointed to the essential atrophic changes in the small intestine, and were similar to the "withering of the villi" described by FAIRLEY and MACKIE in sprue. In those animals with prolonged diarrhoea there was flattening of the villi with decrease of reticular tissue. Degenerative changes were constantly present in Auerbach's plexus; in most cases it was enlarged, oedematous and easily recognized. In the nervous system slight degeneration was evident in the posterior columns and nerve roots: occasionally there were similar changes in the lateral columns.

Haematological studies revealed mostly hypochromic anaemia; in a few it was markedly microcytic. In not a single case was macrocytosis observed. Lesions of the large intestine were evidently the result of superimposed bacillary dysentery and in one instance *Bact. dysenteriae* (Flexner) was isolated.

The intestinal changes could not be ascribed to deficiency of any particular vitamin, but were closely similar to those described by MCKENZIE in nutritional diarrhoea in E. African natives [this *Bulletin*, 1940, Vol. 37, p. 809]. *P. Manson-Bahr.*

BATRA (Brij Lal). **Pellagra in Bilaspur State.**—*Indian Med. Gaz.* 1942. May. Vol. 77. No. 5. pp. 269–271.

Twenty cases of pellagra are reported in Bilaspur State which is situated in the Simla Hills. Four of them were considered to be typical, while the remaining cases were less severe and were classed as atypical. Of the whole group of patients fourteen were male, and all were Hindus of the poorer classes, living on a staple diet of maize and dâl. Clinical improvement followed the administration of a well-balanced diet with nicotinic acid and marmite. *L. J. Davis.*

JEGHERS (Harold). **Nutrition: the Appearance of the Tongue as an Index of Nutritional Deficiency.**—*New England Jl. of Med.* 1942. Aug. 6. Vol. 227. No. 6. pp. 221–228. [57 refs.]

It is not possible to summarize this article, which itself is of the nature of a summary. "It is the purpose of this review to point out that the modern physician can use this simple observation [the appearances of the tongue] to a much better advantage than his predecessor."

The author has succeeded in his aim and presents an interesting account of the tongue in general diseases and in diseases of malnutrition; reference is made to the dry tongue, the coated tongue, the glossitis of nicotinic acid and riboflavin deficiency, Hunter's glossitis, Moeller's glossitis, atrophic glossitis, black hairy tongue and wandering rash of the tongue. For all those unfamiliar with these various conditions Jeghers gives an excellent summary with a useful list of references.

H. S. Stannus.

MOORE (Robert A.), SPIES (Tom D.) & COOPER (Zola K.). **Histopathology of the Skin in Pellagra.**—*Arch. Dermat. & Syph.* 1942. July. Vol. 46. No. 1. pp. 100–111. With 5 figs. [18 refs.]

Although a number of histological studies of the skin lesions in pellagra have previously been recorded, little or nothing is known about the histology of clinically unaffected skin in this condition.

This paper discusses the results of histological studies of biopsy specimens from clinically affected and unaffected areas of skin from 15 patients with clinical pellagra. The skin from both areas was hyperkeratotic, while parakeratosis and acanthosis were found only in the actual lesions. Atrophy of the epidermis, oedema and a moderate infiltration of lymphocytes were also found both in the affected and unaffected skin. The authors conclude, since the skin reacts favourably to nicotinic acid treatment, that these changes are to a considerable extent reversible and that they may represent a specific response on the part of the skin to a deficiency of part of the vitamin B complex.

L. J. Davis.

LEONIDA (Josif). Ist Pellagra erblich? [**Is Pellagra Hereditary?**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1940. July. Vol. 44. No. 7. pp. 316–319.

This article is noticed here for two reasons: First, because it has been thought worthy of acceptance by a reputable journal; second, because any new theory as to the aetiology of a disease whose causation is still obscure should be given at least a hearing.

The author quotes four cases, or rather family groups of cases, which have come under his observation:—

1. A child, 8½ years old, "typically pellagrous" since 1936; the father became pellagrous three years later at age 39. Therefore, "the pellagra factor was present in the germ cells of the father at a time when the pellagra was not yet manifest." A paternal uncle of the child became pellagrous in 1937 and died of the disease. "Here," says the author, "the heredity is obvious." [A strange interpretation of the facts of heredity; the order of appearance of the pellagra was first in a child born in 1931 and developing the disease at the age of 5 years; next, in an uncle becoming pellagrous the following year at age 30 odd, and lastly, in the father two years later still.]

2. A woman of 44 years, her mother, a sister, a first cousin (a woman also of 44 years), her mother and two daughters (of five children) were all pellagrous. The author's comment on this series is: "Heredity showed itself in the females only and this rules out any avitaminosis"—an obvious *non sequitur*.

3. A woman of 35 years with "latent syphilis." Pellagra symptoms appeared in 1936 and were "cured with ovarian preparations." Relapses in each of the following three years were treated with folliculin. The next year one of her children, aged 4 years, a congenital syphilitic, showed symptoms of pellagra. Inherited syphilis, says the author, is of the utmost significance in leading to photosensitiveness of the skin. "The pellagrous erythema in this case [presumably the mother] can be controlled by male or female sex hormone without the giving of vitamins, improving the nutrition or protecting from sunlight." [Is the rash pellagrous at all? May it not be syphilitic?]

4. A woman of 43, with "typical pellagra and acute mania with hallucinations and delirium." She was given three injections of 10,000 units folliculin, 12 injections of bismuth and 6 gm. of neosalvarsan. At the conclusion of treatment the "pellagra was completely cured and she has remained three years without relapse." Every year, however, she has a course of bismuth or solusalvarsan injections. [It is difficult to see where the heredity comes in here. The symptoms would be explained by climacteric in a syphilitic.]

The author concludes that pellagra is due to an hereditary constitutional factor and that nutrition and avitaminosis play no part, that what is hereditary is the photosensitivity of the skin and that the intestinal symptoms are purely secondary. "As burns of the skin may be found associated with duodenal ulcer at autopsy, so may pellagra dermatitis, which bears an analogy with burns of the first and second degrees, be associated with gastro-intestinal disturbances." Finally, "it is not logical to treat a gastritis or a fermentative process in order to cure a patient with photosensibility, inherited or acquired; much more hopeful is it to treat the photosensibility with hormones and antisyphilitics." [A most unconvincing paper.] *H. Harold Scott.*

ZSCHUCKE (Johannes). Beitrag zur Kenntnis des Vitaminstoffwechsels beim westafrikanischen Farbigen. [**The Vitamin Metabolism of West African Negroes.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1940. June. Vol. 44. No. 6. pp. 281–289.

The author studied the vitamin C nutrition of nine healthy natives and 35 native hospital patients. Vitamin C was estimated in the urine before and after a test dose of ascorbic acid. It was assumed that saturation exists when 100 cc. of fresh urine reduces more than 5 mgm. of dichlorophenolindophenol. The healthy persons showed a remarkably high vitamin C excretion and even the hospital patients compared favourably with "healthy Germans." Only patients suffering from trypanosomiasis had a very reduced urinary output of ascorbic acid and needed extremely large amounts of vitamin C to maintain an adequate excretion level. The paper contains two tables, one giving the results of vitamin C estimations on a number of tropical plants, and the other the results of provitamin A determinations on some tropical and imported food products. The chemical methods employed were

such that the results can be considered only as approximate. The author was unable to find clinical evidence of any type of avitaminosis in Spanish Guinea. *H. N. Green.*

SPRUE.

PACHECO MARROQUIN (Salvador). Contribución al estudio y tratamiento del sprue. [**Sprue and its Treatment.**]—*Rev. Med. Veracruzana.* 1942. July 1. Vol. 22. No. 7. pp. 3725–3744. [Bibliography.]

This article is very unequal. Some of it partakes of the nature of a clinical lecture and views are stated without any attempt to discuss their relative values, such as, for example, that sprue is thought by some authorities to be an infective disease, by others attributed to dysentery, fistula, abscess or enteric fever, or again that it may be a complication of haemorrhoids, syphilis or tuberculosis. Another peculiarity is the author's classification of blood cells; normoblasts, megaloblasts and monoblasts [whatever these may be—the term is not defined] are all classified as leucocytes.

The main interest, however, lies in yet another theory of causation and a mode of treatment based thereon. The author's view is that tropical and non-tropical sprue are one and the same and are due to disturbance of the bone-marrow which in turn destroys endocrine synergy. As a consequence the deficiency or absence of hormones leads to mal-assimilation of certain vitamins and so to avitaminosis and non-assimilation of fats. Parathyroid dysfunction causes loss of fixation of calcium in the blood with resultant reduction of alkalinity and change in the *p*H. Acidity favours the development of monilia, etc., while the endocrine disturbances interfere with or alter the functioning of the sympathetic which regulates digestion and results in functional and trophic disorders. "On correcting the disturbance of the bone marrow, the endocrine synergy recovers and the symptoms of sprue disappear," hence the treatment of sprue is to be based on injections of extract of bone marrow, strengthened by liver extract, hormones (pancreas, parathyroid, etc.) and vitamins. [This seems quite simple and the only thing lacking is a fact or two to support the theory.]

The author gives detailed results of the treatment of 53 cases of sprue; these are classified as follows:—

1. Six treated exclusively with vitamins, B_1, B_2 complex, C and K according to the indications. Improved but relapsed.

2. Five treated exclusively with "antithyroid extract" [? parathyroid]. Improved, but serious relapses.

3. Seven treated with hormones: parathyroid extract, suprarenal cortex and pancreas. Improved but relapsed.

4. Six treated exclusively with liver. Much improved, but relapsed after long intervals.

5. Six treated solely with bone marrow. The author's method is to take the marrow from the sternum of a healthy person and inject it into the medulla of the sternum of the patient [no dosaage stated, nor intervals between injections]. All six cured, but without complete restoration of the total red cell count.

6. Fifteen treated with the marrow and with liver. Cured without relapse and with complete restoration of the blood.

7. Five treated with marrow, liver, parathyroid and suprarenal. Results as in the last, but attained more quickly.

8. Five who died in spite of all the above methods of treatment.

According to the above, groups 5, 6 and 7 were the only ones cured, 26 patients in all, but in a table of final results the author shows that three died, three improved but had serious relapses, and 47 were "completely cured with no relapses up to the present." [No attempt is made or explanation offered to reconcile these discrepancies.]

H. Harold Scott.

HAEMATOLOGY.

JONES (E. Baker). **Sub-Clinical Anaemia of School-Children in Southern Rhodesia.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1942. Aug. 31. Vol. 36. No. 2. pp. 99–116. With 2 diagrams. [11 refs.]

A preliminary survey having suggested that sub-clinical anaemia was the rule rather than the exception among school-children in Southern Rhodesia, careful determinations of the haemoglobin levels in 2,173 European and 304 coloured school-children were made by Newcomer's method. In the absence of a normal standard for healthy persons living under ideal conditions in Southern Rhodesia, the average figure for the European children was taken as the normal standard and was found to be 13·49 grammes of haemoglobin per 100 cc. blood (equivalent to 97·7 per cent. on the Haldane scale). The lower level for normality was arbitrarily placed at 90 per cent. of this figure, to the nearest 0·5 gramme of haemoglobin, namely at 12 grammes haemoglobin per 100 cc. blood (87 per cent. Haldane), any figure below this being taken as representing anaemia.

Of the European children 320 (14·7 per cent.) were found to be anaemic, and of the coloured children, whose average level was only 12·99 gm. haemoglobin per 100 cc. blood, 70 (23 per cent.) had values below 12 gm. and were therefore considered to be anaemic.

It has been said that the salient anaemia-producing influences in Southern Rhodesia were disease [presumably parasitization], climate, diet and altitude. Of the European children 11·5 per cent. harboured parasites, the average haemoglobin values for these children being 13·2 gm. per 100 cc. blood, and 23·2 per cent. of the children had values below 12 gm. Similarly 13·5 per cent. of the coloured children were parasitized, their average haemoglobin value being 12·08 gm. per 100 cc. blood, and 36·6 per cent. of them were anaemic. Analysis of determinations made during the warm season lent no support to the theory that anaemia may be dependent on climatic factors, and no evidence was obtained that the altitude of the plateau region of the Colony had any effect in raising the haemoglobin index. A comparison was made between the boarders in school hostels and the non-boarders, with a view to examining the effects of diet on the haemoglobin levels, but owing to the conditions of the observations no clear results were obtained, although boarders receiving a relatively high protein and

iron intake showed less anaemia than those receiving a low intake; incidentally, parasitic infections among the European boarders did not appear to have any marked effect on the haemoglobin concentration of the blood. There was, as is commonly found in children, a gradual increase in the haemoglobin levels with increasing age, possibly due to increasing immunity to various infections and to an increasing proportion of protective foods in the diet. *F. Murgatroyd.*

VENOMS AND ANTIVENENES.

Chopra (R. N.), Chowhan (J. S.) & Chopra (I. C.). **Sterilization of Snake Venom Preparations.**—*Indian Med. Gaz.* 1942. Jan. Vol. 77. No. 1. pp. 23–26. [10 refs.]

Snake venom preparations are often contaminated, and it is important, in view of the therapeutic use of these venoms now being made, that a method of sterilization should be found. It is known that heat destroys certain properties of venom, for instance, cobra venom heated to 75°C. loses its haemorrhagic principle. Filtration through Seitz discs also removes certain principles.

The authors have investigated this subject, using the venoms of the cobra and *V. russellii*. Tyndallization (heating to 60°C. for one hour daily on three successive days) is unsatisfactory for Russell viper venom, organisms being found after the process; this venom is often contaminated even in the dry state. Filtration is satisfactory from the point of view of sterilization, but affects the toxicity and therapeutic efficiency. Sterilization by the addition of sodium merthiolate (0·01 per cent. final concentration), phenol (0·25–0·5 per cent.) or tricresol (0·25–0·5 per cent.) is effective and maintains intact all the active principles.

Care should, however, be taken in the collection of venom. The authors point out that the buccal cavities of snakes kept in well-drained, open parks are usually relatively sterile, but that in captivity or after frequent extraction of venom, the bacterial flora is much increased. Aerobic and anaerobic organisms may be found. *C. W.*

Boquet (Paul) & Delaunay (Albert). Réactions du mésentère du cobaye au venin et à l'anavenin de *Vipera aspis*. [**Reactions of the Guineapig Mesentery to the Venom and the Anavenene of** *V. aspis*.]—*C. R. Soc. Biol.* 1942. Apr. Vol. 136. No. 7–8. pp. 320–321.

Venom injected into the peritoneal cavity of guineapigs produces in the mesentery numerous and characteristic ruptures of the blood vessels. The capillaries are dilated and the endothelial cells swollen and globular. Leucocytes enter the mesenteric tissue; first polymorphonuclears and later mononuclears predominate. Control animals which receive saline injections show no such changes. Anavenene prepared from the same venom, by mixture with formaldehyde, does not give this reaction, nor does a mixture of venom and its anti-serum, though in the latter case there is considerable leucocyte reaction such as occurs after injection of immune or normal serum.

When venom loses its toxicity under the influence of formaldehyde, therefore, it loses at the same time its local haemorrhagic power, and this action, like the other toxic and diastatic properties, is inhibited by antivenene. *C. W.*

Brooks (Georges). Sur l'étude chimique et spectrographique de la fluorescence des venins de serpents. [**Chemical and Spectrographic Study of Fluorescence of Snake Venoms.**]—*Ann. Inst. Pasteur.* 1940. June. Vol. 64. No. 6. pp. 558–564. With 1 fig. & 2 charts.

Both viper and cobra venoms fluoresce when examined in ultraviolet light with a microfluoroscope ; the fluorescence is bluish, but with viper venom there is a golden yellow tint. The fluorescent substance has certain properties which relate it to the synthetic flavines (riboflavine), and while in viper venom this exists in the free state, in cobra venom it is combined with protein: Fluorescence has not previously been described in snake venom, but it is possible that the substances responsible for it may have some physiological action.

The work was done on venoms of *Vipera aspis* and *Naja tripudians*. *C. W.*

Vellard (J.). Modifications sanguines provoquées par les venins (quatrième mémoire). Action hémolytique et variations de la résistance globulaire *in vivo*. [**Haemolytic Action of Snake Venoms, and Variations of Erythrocyte Resistance** *in vivo*.]—*Ann. Inst. Pasteur.* 1940. Sept. Vol. 65. No. 3. pp. 170–197. With 6 graphs.

[For previous papers in this series see this *Bulletin*, 1934, Vol. 31, p. 101 ; 1935, Vol. 32, pp. 376, 380 ; 1936, Vol. 33, p. 390.]

The haemolytic action of venoms has been extensively studied *in vitro*, but the *in vivo* action is little understood. The phosphatidase activity of venom is exercised on red cells and on the plasma, and plays a large part in haemolysis, but the venoms of the Viperidae and some of the Colubridae possess coagulant and protease principles which tend to prevent haemolysis and to increase red cell resistance. In the present research the authors have found that the venom of *Naja tripudians* (which possesses high phosphatidase properties but is non-coagulant) provokes, in the blood of injected dogs, the appearance of powerful haemolytic activity which leads to massive destruction of red cells and release of haemoglobin. In this positive phase there is diminished resistance of the cells to hypotonic solutions, but this phase is always short. In the later (negative) phase the breaking down of phosphatide is increased, but the haemolytic power disappears and cell resistance to hypotonic saline returns to normal. Natural haemolysins for foreign red cells (sheep, horse) are diminished in the positive phase but soon return to normal.

The diminution of resistance to hypotonic solutions during the positive phase is due to haemolysins formed in the circulation. The venom exercises an action on the cells, which are increased in size and which lose resistance to the haemolysin of venom ; but in the negative phase this resistance is increased. Autohaemolysins rarely persist for more than one hour after injection ; regeneration of phosphatides is slow and it is usually 24 hours before the serum recovers its haemolytic power in the presence of a venom *in vitro*.

Venoms which are phosphatidasic and coagulant (*Crotalus terrificus*) have much the same effect, but the coagulant action strengthens cell resistance to venom haemolysins.

With some Crotalines the *in vivo* haemolytic action may be completely masked, and cell resistance to hypertonic solutions may be markedly raised from the first, but haemolytic property reappears if these venoms are heated at 72°C. to destroy protease.

The rapid breaking down of blood phosphatides, which causes rapid disappearance of the haemolytic property of plasma, and the rapid increase in cell resistance to the venom haemolysins, prevent cell destruction and play an important part in the defence of the organism. *C. W.*

Ramon (G.), Boquet (P.), Richou (R.) & Nicol (L.). Les anavenins spécifiques et les substances adjuvantes et stimulantes de l'immunité dans la production des sérums antivenimeux respectivement dirigés contre les venins de *Cerastes cornutus* et de *Naja-haje*. [**Anavenenes and Adjuvants Stimulating Immunity against the Venoms of** *C. cornutus* **and** *N. haje*.]—*Ann. Inst. Pasteur.* 1941. Nov. Vol. 67. No. 5. pp. 355–358.

The authors refer to the reduction in time effected, in the production of high titre antitoxins, by the use of anatoxins of diphtheria and tetanus which are mixed, before injection, with tapioca. This substance acts apparently as a stimulant in the production of immune bodies. They now record the application of their method in connexion with the preparation of antivenenes against the poisons of *C. cornutus* and *N. haje*. The respective anavenenes, prepared by the well-known method were divided before each injection into two parts, to one of which was added tapioca, to the other tannin, but in proportions not detailed. The injections were made every few days in horses, and the titres of the sera estimated at intervals. In this way potent antivenenes were obtained in 4–5 weeks, whereas by using the old method of injecting crude venom, immunization for 8–10 months was necessary. *C. W.*

TROPICAL OPHTHALMOLOGY.

A Review of Recent Articles.—XL.*

Eyelids.—Pathan[1] has described an *operation for trichiasis* which he has found satisfactory in Karachi. It consists in the grafting of a skin-flap taken from the surface of the lid into an incision made in the inter-marginal space. The centre of the elliptical skin-flap is left attached and its two extremities are drawn through a tunnel made in the lid border between the wound in the surface of the lid and that in the inter-marginal space. The ends of the flap are then spread out in the inter-marginal wound and stitched in place. The author

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For the 39th of this Series see Vol. 39, pp. 401–404.

[1] Pathan (H. A. H.). Trans-grafting Operation for Trichiasis and Entropion of the Upper Lid.—*Indian Med. Gaz.* 1942. Apr. Vol. 77. No. 4. pp. 204–206. With 7 figs.

states that he has found van Milligan's operation and its modifications, practised successfully by many ophthalmic surgeons, somewhat unsatisfactory.

Conjunctiva.—THYGESON & STONE[2] have studied 50 cases of *inclusion conjunctivitis* occurring in infants, children and adults, and conclude that the infection is mostly derived from a low-grade urethritis in the male and a sub-clinical cervicitis in the female, limited in the latter case to the region of transitional epithelium just within the external *os uteri*. Gonorrhoea and the virus disease are often associated, but appear to be entirely independent infections. Infection of infants commonly occurs during delivery, and adult infection by way of swimming pools and direct contamination. The disease is stated usually to be rapidly cured by sulphanilamide or a derivative.

Trachoma.—WILSON,[3] in a review of the pathology of *trachoma*, remarks that in Egypt one seldom sees a case of uncomplicated trachoma from the earliest time of infection, since practically all patients have suffered from a previous Koch-Weeks or gonococcal conjunctivitis or commence with an acute mixed infection. In observed cases of accidental infection the disease has always commenced a few days after infection with moderately acute symptoms, but the onset is generally insidious. His summary is that " the pathology of trachoma is that of a chronic inflammatory disease which is entirely independent of any so-called constitutional lymphatic dyscrasia. The type of pathological reaction suggests a virus infection and this is probably represented by the elementary granules of the Prowazek-Halberstaedter body. Papillary hypertrophy, follicle formation on the tarsal conjunctiva and corneal lesions invariably occur, and the disease terminates in scar formation."

Struck by the contradictory reports regarding the value of sulphonamide therapy in trachoma, JULIANELLE & SMITH[4] have conducted experiments on the monkey (*M. rhesus*) to test the value of the drug when used *in vitro* on infective trachomatous material. They state that it was not possible to demonstrate that sulphanilamide has any appreciable effect on the infective capacity of the virus of trachoma. Analysing the result of sulphanilamide therapy during a three years' trial in the treatment of 338 white trachomatous patients in Kentucky, SORY[5] has concluded that the drug only exceptionally arrests trachoma and is, by itself, less reliable than other accepted forms of treatment, but that it certainly accelerates recovery when employed in combination with other therapeutic measures ; even then recurrence may take place, thus proving that the disease has been arrested but not cured.

Observers in Tunis have reported remarkably good results in the treatment of the disease by the oral administration of a preparation

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[2] THYGESON (Phillips) & STONE (William), Jr. Epidemiology of Inclusion Conjunctivitis.—*Arch. Ophthalmology*. 1942. Jan. Vol. 27. No. 1. pp. 91–119. With 5 figs. [35 refs.]

[3] WILSON (Rowland P.). The Pathology of Trachoma.—*Acta Ophthalmologica Orientalia*. Jerusalem. 1940. Jan. Vol. 2. No. 1. pp. 1–44. With 28 figs. [10 refs.]

[4] JULIANELLE (L. A.) & SMITH (J. E.). Studies on the Infectivity of Trachoma. XI. The Effect of Sulfanilamide on the Virus.—*Amer. Jl. Ophthalm.* 1942. Mar. Vol. 25. No. 3. pp. 317–321.

[5] SORY (Robert). Sulfanilamide in the Treatment of Trachoma. Results after Three Years of Trial.—*Amer. Jl. Ophthalm.* 1942. June. Vol. 25. No. 6. pp. 713–720. [19 refs.]

called azoique 33 (or G.33). This is a potassium and sodium salt of parasulphamido-phenyl azo salicylic acid whose chemical formula is stated to be

and forms a tasteless, insoluble, yellow powder. BARRAT[6] has used it in doses of 1·5 gm. given every morning and evening for a week. The drug is omitted during the following week and treatment is then resumed. This observer and BERGE[7] claim that corneal complications are nearly always rapidly relieved, that secondary infections are controlled and that follicle absorption is effected. Chronic cases require treatment for two and a half to three months in order to effect a cure. No injurious blood changes occur even when large doses are given, but a slight leucopenia followed by a moderate lymphocytosis was seen in a few patients. The same two observers[8] have used a soluble lithium salt of the drug to test its toxic action when administered to the rabbit intravenously and have found that very large doses produced no harmful effect. They suggest that intramuscular injection of the drug might prove useful in the human subject.

ESTIBOTTE and ROUSSEL[9] found, in the treatment of 16 school-children, that corneal complications yielded to the drug, but that lesions of the tarsal conjunctiva were more resistant. Five of the children failed to respond to the treatment. SÉDAN and others[10] also report favourably, but say that the preparation had no specific action against secondary conjunctival infections, particularly Koch-Weeks inflammation, and that these required suitable local treatment. They noted the occurrence of a definite polynuclear leucocytosis in those cases which responded well to treatment by the drug. BURNET, CUÉNOD and NATAF[11] claim that the drug is practically innocuous and that by its use a clinical cure can be effected in a certain number of cases, and also note the occurrence of a polynuclear leucocytosis in those patients who readily respond to treatment. They have been able to observe the late result in 23 patients and report that in 18 of these a clinical cure had been obtained. It may be noted that most of these required from five to ten months of treatment. GRADLE[12] has

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[6] BARRAT (P.). Essai de traitement collectif du trachome par un composé azoique sulfamidé (G.33).—*Arch. Inst. Pasteur de Tunis.* 1941. Dec. Vol. 30. No. 3–4. pp. 237–244.

[7] BARRAT (P.) & BERGE (Ch.). Recherches sur le traitement du trachome par l'azoique sulfamidé 33. Étude du sang des sujets traités.—*Arch. Inst. Pasteur de Tunis.* 1940. Dec. Vol. 29. No. 4. pp. 409–436.

[8] BERGE (Ch.) & BARRAT (P.). Quel est le degré de toxicité chronique de l'azoique 33 administré par voie intra-veineuse chez le lapin.—*Arch. Inst. Pasteur de Tunis.* 1940. Dec. Vol. 29. No. 4. pp. 437–449.

[9] ESTIBOTTE (R.) & ROUSSEL (H.). Essai de traitement du trachome avec le composé 33 sur 15 écoliers de la banlieue N. de Tunis.—*Arch. Inst. Pasteur de Tunis.* 1940. Dec. Vol. 29. No. 4. pp. 450–454.

[10] SÉDAN (Jean), KOUTSEFF (A.), LIVON (J.) & GAURAN (M.). Traitement du trachome par le G.33. Étude hématologique.—*Arch. Inst. Pasteur de Tunis.* 1940. Dec. Vol. 29. No. 4. pp. 398–408. With 2 figs.

[11] BURNET (Ét.), CUÉNOD (A.) & NATAF (R.). Traitement chronique du trachome par l'azoique sulfamidé 33. Résultats éloignés. Guérisons.—*Arch. Inst. Pasteur de Tunis.* 1940. Dec. Vol. 29. No. 4. pp. 389–397.

[12] GRADLE (Harry S.). Visual Results in the Trachoma Clinics of Southern Illinois.—*Jl. Amer. Med. Assoc.* 1942. July 4. Vol. 119. No. 10. p. 792.

compared the visual results in 328 trachomatous patients in Illinois treated with sulphanilamide with those in 493 patients treated in other ways. He found that in the former group twice as many showed a definite improvement in their vision and less than half as many showed a deterioration.

Cornea.—Thornwall DAVIS[13] has described his experience of *superficial punctate parenchymatous keratitis* observed in 195 patients in America during the past 13 years. Mild or severe conjunctivitis in association with photophobia, lachrymation and conjunctival injection, especially affecting the bulbar portion, was the common characteristic. A sensation of a foreign body in the eye was experienced. Oedema of the upper lid and ptosis were occasionally met with. Careful examination of the cornea showed minute white flecks in the membrane; wavy "glass lines" too were often observed. These opacities lay in the superficial layers of the cornea and did not involve the epithelium. Davis emphasizes this point and describes the disease as parenchymatous for this reason. Local treatment with phenacine and epinephrine and the use of the Shahan thermophore effected a rapid cure. Bacterial investigations all gave negative results. The author has furnished a comprehensive review of similar conditions met with elsewhere.

Glaucoma.—In a review of the aetiology and treatment of glaucoma DAYAL[14] stresses the importance of the vascular element in the causation of the disease and enumerates the factors which contribute to venous stasis and vasomotor disturbance. For acute congestive glaucoma he recommends a "trap-door iridectomy." This is an operation which differs from the ordinary corneo-scleral trephining in that the trephined disc is replaced after performing iridectomy. For chronic forms of the disease he considers various conservative methods of treatment, but unconditionally advises operation if loss of visual field occurs. He regards Elliot's operation as somewhat uncertain in its results and describes iridencleisis and Lagrange's operation. Cyclodiathermy is another operative procedure recommended by him. For glaucoma secondary to uveitis he performs iridectomy and for that following cataract extraction and discission he recommends cyclodialysis.

DODDS[15] has reported his experience of eye disease met with in an eye clinic at Lagos and based on the observation of 801 patients. Conjunctival disease heads the list with 206 admissions, errors of refraction take second place with 189, and "defective vision" third with 170. Cataract accounted for 58 admissions. Trachoma, spring catarrh and purulent ophthalmia were found to be the commonest conjunctival affections; 66 cases of trachoma were seen and the author found sulphonamides remarkably effective in their treatment. Entropion and trichiasis were seldom seen. Admissions for myopia and astigmatism were equal in number and those for hypermetropia slightly exceeded them. Asthenopia and vitamin B_2 deficiency appear to be

[13] DAVIS (William Thornwall). Superficial Punctate Parenchymatous Keratitis. —*Arch. Ophthalmology.* 1942. Feb. Vol. 27. No. 2. pp. 279–288. [15 refs.]

[14] DAYAL (Sukh.) Aetiology of Glaucoma, its Various Methods of Treatment and their Merits.—*Indian Med. Gaz.* 1942. Apr. Vol. 77. No. 4. pp. 233-240. [13 refs.]

[15] DODDS (G. E.). A Report on an Analysis of Cases at an Eye Clinic in Lagos, Nigeria.—*Brit. Jl. Ophthalm.* 1942. June. Vol. 26. No. 6. pp. 257–265. [17 refs.]

responsible for the majority of the cases of "defective vision," and cases of advanced vitamin deficiency were associated with a pallor of the temporal half of the optic disc. *H. Kirkpatrick.*

DE SILVA (K. J. L.). **The Problem of Trachoma in Ceylon. With a Plea for its Suppression.**—*Jl. Ceylon Branch Brit. Med. Assoc.* 1941. Dec. Vol. 38. No. 4. pp. 346–361. With 3 figs. on 1 plate. [Summary appears also in *Bulletin of Hygiene.*]

No detailed survey of the incidence of trachoma in the central province of Ceylon has as yet been carried out. For the period January 1939 to May 1941, 50 cases were seen at the Eye Institute, Kandy, whilst in Akurana, a village predominantly Muslim, 17 cases were found amongst 636 persons examined. The author believes that trachoma is spread into Ceylon by infected immigrants and that the bazaar areas of the larger towns are the sources of propagation. A series of public health measures on the lines adopted by other countries is suggested. *Arnold Sorsby.*

GOMES (Luis de Salles) & JORDÃO (Sylvio). Sobre a reacção de Weil-Felix no trachoma. [**The Weil-Felix Reaction in Trachoma.**]—*Brasil-Medico.* 1940. Mar. 9. Vol. 54. No. 10. pp. 151–159. English summary. [Summary appears also in *Bulletin of Hygiene.*]

The author tested the sera of 235 patients with trachoma in various stages for the Weil-Felix reaction with *Proteus OXK*, *P. OX19* and *P. OXL* and, as controls, of 30 normal persons giving no history of either trachoma or typhus infection. The cases were classified according to MacCallan's clinical stages. Of these 18 were stage I, 136 stage II (2 of these were verging on stage III), 69 of III and 12 of IV. Altogether 42·3 per cent. were positive in 1 : 200 dilution or more, and of those in stages II and III 42·7 per cent. Of the normal sera 20 agglutinated *Proteus OXK* in dilutions up to 1 : 100 ; hence anything under 1 : 200 should, in the author's opinion, be disregarded.

As regards the individual organisms : *Proteus OXK* was agglutinated in 38·9 per cent. of all the cases and in 38·3 per cent. of those in stages II and III ; *Proteus OX19* in 15·2 and 15·5 per cent. respectively, and *Proteus OXL* in 0·9 per cent. for those in stages II and III or in all four. Seven agglutinated *Proteus OXK* in a dilution of 1 : 400, two *Proteus OX19* in the same titre ; none agglutinated *Proteus OXL* in as high a titre as this.

Clearly, therefore, the reaction is not a dependable one for the diagnosis of trachoma in any stage. *H. Harold Scott.*

MISCELLANEOUS.

MRUGOWSKY (J.). Die Seuchenlage im europäischen Russland. [**The Epidemic Diseases of European Russia.**]—*Med. Klin.* 1942. July 3. Vol. 38. No. 27. pp. 625–629. [Summary appears also in *Bulletin of Hygiene.*]

The author claims to have obtained his information from official Russian reports ; he gives no references to original papers in the

Russian literature. Diseases are grouped under four headings :— (1) Diseases of the respiratory system. (2) Diseases of the alimentary tract. (3) Diseases spread by direct contact. (4) Diseases transmitted by animals [including insects].

In the first group, scarlet fever and diphtheria are common and widespread. Smallpox occurs in all areas, but is most frequently found in the North-east, the middle Volga region and in the Urals. All these diseases are especially prevalent in winter.

Of the alimentary diseases, typhoid and the paratyphoids are the most common. Typhoid is endemic, and is more often found than paratyphoid B ; paratyphoid A, however, is more widespread in the south, particularly between the Caspian Sea and the Don, in the Ukraine and in the Caucasus. Late summer is the season of maximum prevalence of these diseases. Bacillary dysentery is widespread, but whereas in former years the Shiga-Kruse form was common, the type recently found, especially in 1941, has been that due to Flexner Y and E [presumably Kruse-Sonne]. In the Ukraine the heavy incidence was due to the Shiga-Kruse bacillus. The Baltic States have been relatively free, but in White Russia the incidence is considerable ; high figures are reported from Karelia, the Crimea, the Caucasus, Daghestan; the Ukraine, Trans-Caucasia and Uzbekistan. The disease is at its height in summer. Amoebic dysentery is practically absent from the Ukraine, but is not rare in the Caucasus. Intestinal protozoa are found in profusion in Azerbaijan.

Cholera is not endemic, and has not been reported since 1927. Earlier than that, however, it had been found in the Ukraine, and had spread to Poland and Germany ; the possibility of its reappearance must be reckoned with, especially round the Black Sea and the Caspian.

Of the diseases spread by direct contact, trachoma is one of the most common, from the Baltic to the Black Sea. Leprosy is important only in the three Baltic States, and in these it is gradually dying out. Syphilis is no more common in Western Russia than in the rest of Europe, but is more prevalent in the Volga region. The incidence of gonorrhoea resembles that elsewhere in Europe.

Of the diseases transmitted by insects, malaria is the most important. In general, benign tertian is the common form, but in the south there is an area of subtertian malaria, bounded to the north by a line between Kieff, Voronezh, Saratof, Samara and Orenburg. Malaria is especially common along the coasts of the Black Sea and the Caspian, in the country between these seas, and in the valleys of the Dnieper, Besna, Bug, Ingoletz, Don, Donetz and the Volga. There is a focus to the east of Moscow, and the disease is found in the marshy country throughout. Quartan malaria is rare in the Ukraine, but is more common in the Caucasus and the Volga basin. There is a malaria station as far north as the White Sea, near Archangel ; in Karelia there are mosquitoes (Culex and Aëdes) but no Anopheles.

The Russians have created a strong organization for the prevention and treatment of malaria, with institutes at Moscow, Kieff, Kharkov, Dniepopetrovsk, Odessa, Tiflis, Baku and other places. The vectors in the Ukraine are *A. maculipennis messeae* and *atroparvus*, *A. bifurcatus* and *A. hyrcanus* ; in the Caucasus *A. superpictus*, *A. plumbeus*, and *A. pulcherrimus* are implicated. Blackwater fever is rare in the Ukraine, but common in the Caucasus.

Aëdes aegypti is found in the Caucasus ; yellow fever is therefore theoretically possible there.

Typhus is the most prevalent disease after malaria and typhoid ; it is louse-borne and is accompanied in places by relapsing fever and trench fever. The greatest incidence of typhus takes place in White Russia and, to the east of Moscow, throughout the area from the White Sea to the mouth of the Volga. The Baltic States are relatively free, as are the Ukraine and the Caucasus. The maximum prevalence of typhus is usually found between January and March, but cases are reported throughout the year.

Sandfly fever is widespread in the Caucasus ; it affects chiefly new-comers and the indigenous children. The sandflies are found in the Crimea and along the shore of the Caspian, but not on the Caucasian coast of the Black Sea.

Plague is associated with ground squirrels and rats ; it is well known in the regions of the Lower Volga, North Caucasus and the Don near Rostov. These are old foci, and the disease has not recently spread from them. Tularaemia is common in the south-east, especially in spring when the water-rat holes are swamped in the floods, and the animals are caught for their skins.

Anthrax is fairly common in the cattle areas, especially in the form of a mild pustule. *C. W.*

KARK (Sidney L.). **A Health Service among the Rural Bantu.**—*South African Med. Jl.* 1942. May 23. Vol. 16. No. 10. pp. 197–198.

In this short paper are brought out several points of great interest and importance to those who attempt to raise the standard of health in African natives. It is emphasized that the provision of curative medical facilities is at present insufficient to meet the needs of the Native Territories of the Union of South Africa, and, moreover, that curative services, which do not attempt to prevent or modify the basic causes of the important diseases, must be brought within the sphere of a broader health scheme. On the other hand a purely preventive service would fail to impress itself on a people whose conception of a doctor is of one who can cure. Health instruction and curative medicine must therefore be combined, and were so combined in the Health Unit formed by the author at Polela.

The people among whom the work was carried on fail to grow all their own food requirements, and exchange their labour for the goods they receive. They are as ignorant and conservative as other native communities and they suffer from the usual diseases, helminthic infections, dysentery, infant diarrhoea, typhoid fever and venereal disease ; they also show evidence of malnutrition.

The Unit undertakes first to get to know the people by house visits. These routine visits, unconnected with temporary illness, are considered more important than follow-up visits after disease, which may quickly be forgotten once the disease is ended. A school health service has been instituted, with clinical examinations of the children, supervision of sanitary arrangements, and the provision of school meals. The native teachers are ignorant of hygiene, but special courses of instruction have proved successful in stimulating their interest. It is realized that school work alone will not succeed unless the parents understand the general principles, and that, therefore, the house visits are essential. Clinics are held for curative purposes, special attention being paid to venereal disease.

Work is carried out in conjunction with the Agricultural Department, and a model vegetable garden, with compost pit and including simple anti-erosion measures, has been instituted in the middle of the community. This is an important demonstration [and it may be expected that the common African faculty for imitation may help] but the author shows that he is fully aware of the fact that for success there must be cooperation between the Magistrate, Medical Department, education authorities, missionary bodies and teachers.

C. W.

Hueck (Otto). Krankheitsverteilung in einem ländlichen Bezirk der Kwangtungprovinz. [**Diseases of a Rural District of Kwang Tung Province of China.**]—*Deut. Trop. Ztschr.* 1941. July 15. Vol. 45. No. 14. pp. 427–436. [10 refs.]

The author gives records of 7,000 patients seen at his mission hospital in Tung Kung during a period of 8 years ; the population was occupied chiefly in agricultural pursuits. The principal diseases were as follows : Entropion and trachoma were very prevalent. Pulmonary tuberculosis was found, especially in school teachers, but also among the peasants ; non-pulmonary tuberculosis, of glands, bones and joints, was not uncommon. Malaria is not very prevalent, and the principal form is benign tertian, though subtertian exists. [Nothing is said of seasonal periodicity or of the common vectors.] Kala azar was not seen. Infection with *Clonorchis sinensis* was not rare, and was more often found in the more wealthy town dwellers than in the peasants. Ascaris infection is widespread and hookworm disease is common in the peasants.

Typhoid is endemic but cholera has not been seen recently ; plague has not appeared for many years. Leprosy, more a disease of country than of town dwellers, is often observed, but its prevalence is hard to estimate.

Beriberi was diagnosed in a number of cases. Cirrhosis of the liver was fairly common ; its aetiology is obscure and it is not, apparently, connected with the presence of *C. sinensis*. Venereal diseases are fairly prevalent in the towns. Skin diseases of many kinds are rife and ulcer of the leg is frequently seen. The fevers of childhood occur here as in most other countries. *C. W.*

Gleitsmann (Hans-Wilhelm). **Pyomyositis tropica=Pyomyositis trophica ?**—*Arch. f. Schiffs- u. Trop.-Hyg.* 1940. July. Vol. 44. No. 7. pp. 301–315. [35 refs.]

As a clinical description and a general account of pyomyositis this article is good. From this point of view, however, it gives no more information than may be found in the better text-books. The author discusses the correctness of the name of the disease ; according to some, Ziemann and Mense, for example, it is confined to the tropics, according to others it exists in non-tropical countries also. Ruge and de Barres are among the supporters of this view, and Schönbrunner claims to have seen it in Vienna, and Holm in Sweden. Hence there naturally arise the questions : Are these muscle abscesses of the same origin in the tropics as in temperate climates ? Or are they the same even in the different tropical regions where they are met with ? Or,

yet again, is it, as Ziemann stated in 1904, a disease *sui generis*, or a symptom of several, for example the mumu fever of Samoa, a complication of filariasis, or the result of trauma.

These questions are put forward by the author, but are not answered. Apart from the clinical description mentioned, the article is a poor one and not very helpful. As is well known the bacteria present vary: staphylococci, streptococci, Pasteurella being met with, or, in some cases, no organisms at all [Scott's findings (*Jl. Trop. Med. & Hyg.*, 1912, Vol. 15, pp. 97–100) are not referred to in the text or bibliography]. The author discusses, mainly by references to the literature, avitaminosis, chiefly because the disease occurs in rice-eaters and because in beriberi there are specially painful spots along the fibres of tender muscles, and because in some cases the giving of Betaxin seems to cut short the disease. Secondly, allergy, because of the eosinophilia shown by some patients [considering the almost universal existence of helminthic infestation in the tropics, this argument can have little weight, unsupported]. Thirdly, the possibility of its being of trophic nervous origin, because the nervous system has " an immediate influence on nutrition of muscle " and muscle dystrophy may have a nervous origin. Fourth [mentioned here because the author lays considerable stress on it] that it is related to Bornholm disease, epidemic myositis [see *Bull. of Hyg.*, 1942, Vol. 17, p. 330. Surely only an armchair critic could see any similarity between this and muscle abscess. Nobody with practical experience could possibly confuse them].

H. Harold Scott.

v. Bormann (F.), with the technical assistance of J. Hollack & E. Kirchhöfer. Myositis tropica acuta-Staphylokokkensepsis. [**Tropical Myositis an Acute Staphylococcal Infection.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1940. Oct. Vol. 44. No. 10. pp. 458–474. With 3 figs. [20 refs.]

The author's observations are based on a consideration of some of the literature on this subject and on 11 cases seen by him in negroes. He begins with a general clinical description of the average case: fever (to 40°C.), severe pain in the affected muscle with local induration going on to softening and pus formation, and, sometimes, with metastatic collections of pus. Some present themselves in the abscess stage; in other cases the induration recedes without going on to abscess. In the author's experience the pus is greenish-white, thick, often haemorrhagic. The muscles chiefly involved are the glutei, quadriceps femoris, deltoid, biceps, flexors of the forearm, and less often the pectorals and abdominal muscles. The majority of the patients are between 20 and 30 years of age. Four of the author's eleven patients had one abscess only, one had two, four had three, and one as many as ten. One died and autopsy revealed metastases in the muscles and pericardium.

The causation is discussed, mainly by references to the literature of helminthic infestations—*F. bancrofti*, *D. medinensis*, *O. volvulus* and bacterial infection. The author believes the condition to be due to a haemolytic staphylococcus, commonly *aureus*, less often *albus*, but of a peculiar type which, experimentally, does not produce the condition in guineapigs, rats or mice, but does so in rabbits. The site of entry in human cases is uncertain, but spread is probably by the blood stream, for lymphatics and lymph nodes show no signs of inflammation

or enlargement. He quotes RODENWALDT, saying that the portal of entry may be the nose and throat, but adduces no evidence in support of the idea. He concludes by saying that this myositis is limited to warm countries, but that no reason can be given for this limitation, nor for its peculiar localization in the muscle tissues ; that the organism concerned is a Group I staphylococcus such as is common in pyogenic lesions in Europe. [On the whole a rather disappointing article.]

H. Harold Scott.

BOPAIYA (M. S.) & RAO (M. V. Radhakrishna). **Note on "Tropical Ulcer" in Coorg.**—*Indian Med. Gaz.* 1942. Mar. Vol. 77. No. 3. pp. 139–140. [10 refs.]

The prevalence of tropical ulcer in Coorg has not hitherto been recorded in the literature, but during the course of a nutritional survey the authors found many cases, and analyse 400 from two hospitals. The condition, as described, appears to be the usual phagedaenic ulcer so frequently seen throughout the tropics. It was most common in males and in coolies employed on plantations or in men cultivating their own land. Attempts to find leishmania were unsuccessful. Signs of malnutrition, such as phrynoderma, were often present, but the authors point out that tropical sore is not common in coolies in the Nilgiri hills, whose diet is as defective as that of the Coorg coolies. Malaria, hookworm disease and severe anaemia are uncommon in the Nilgiri hills but are common in Coorg, and the authors incline to the view that anaemia and debility resulting from blood-destroying diseases are factors of importance in the aetiology of tropical ulcer, and note that the onset is usually associated with slight or moderate trauma.

C. W.

TAYLOR (Frederic W.). **Chronic Hemolytic Streptococcus Ulcers of the Extremities.**—*Jl. Amer. Med. Assoc.* 1942. Apr. 4. Vol. 118. No. 14. pp. 1196–1199. With 3 figs.

Although chronic haemolytic streptococcal ulcers of the extremities form a clear-cut entity, little attention has been given to them, only five cases being reported in recent literature.

The lesion starts as a slight cutaneous excoriation or insect bite which after a few days forms a small indolent ulcer, usually about 1 cm. in diameter, although it may reach 5–6 cm. Pain is not prominent. There is only a slight tissue reaction confined to the reddened ulcer margin, and surrounding oedema and lymphangitis are absent. The base weeps watery serum from an atrophic granulation tissue. Haemolytic streptococci can be recovered by culture, frequently with contaminants, *Staphylococcus albus* being the commonest. Practically all the ulcers are on the lower leg and they are most frequent in children and young adults, a background of such conditions as old varicosities, arteriosclerosis, syphilis and frost-bite being uncommon.

Although a few of the 31 cases reported by the author healed under local treatment, most had been treated in this way by a variety of agents without success. The response to oral sulphanilamide in adequate dosage, 4·4·5 gm. daily on an average, was striking, although the blood levels of the drug were rather low, varying from 3·5 to 5·2 mgm. per 100 cc. In 19 cases the ulcers healed completely in an average of 1·8 weeks although they had first been treated locally with all

types of drugs for an average of 7·7 weeks; one case failed entirely to respond to sulphanilamide and possibly a fungus was present although it was not demonstrated. Ulcers superimposed on a background of varicosities, phlebitis, arteriosclerosis or syphilis also failed to heal, but sulphonamide may be useful in such cases as an adjunct to other forms of therapy. The local use of sulphonamide might be worth trial, but it was not used in the present series. *F. Murgatroyd.*

HAMBURGER (H. J.). **Sulphonamides in Topical Application.**—*Indian Med. Gaz.* 1942. Mar. Vol. 77. No. 3. pp. 135–139. [23 refs.]

In this paper the author discusses the local treatment of wounds and infected tissues with sulphonamide drugs, and gives a résumé of some of the literature on the subject. He refers to his own experience in the treatment of 120 cases of frontier sore, a condition superficially resembling cutaneous leishmaniasis, but in which leishmania are not found, and of impetigo, varicose ulcers and infected injuries, with details of illustrative cases. Comparison with other methods of treatment is not given in detail, but the general impression is that response to the sulphonamide treatment is good and often dramatic. Early trachoma responds well to local application of sulphonamide to the conjunctiva, but when advanced does not respond; cutaneous leishmaniasis shows no improvement under this treatment.

The preparations used were 6 per cent. soluseptasine ointment, prontosil soluble 1 to 5 per cent., prontosil base ointment 5 per cent., or powdered drugs either as powder or incorporated in glycerin for paints or in petrolatum for ointments. For ophthalmic work the finest ointment-bases should be used and the sulphonamide should be in a strength of not more than 1 per cent.

[For the author's previous work on frontier sore, see this *Bulletin*, 1940, Vol. 37, pp. 223, 812.] *C. W.*

MUWAZI (E. M. K.) & TROWELL (H. C.), in collaboration with R. S. F. HENNESSEY. **Liver Diseases and Jaundice in Natives of Uganda.**—*East African Med. Jl.* 1942. May. Vol. 19. No. 2. pp. 40–66. [10 refs.]

[The bulk of the work reported in this paper was done by the first-named author, who also prepared the first draft. It is pleasant to realize that the African Assistant Medical Officers are engaged not only on routine medical work, but also on original scientific investigations.] The authors report 71 cases of liver disease or jaundice treated in their wards in one year, but exclude from consideration cases of secondary infective hepatitis, such as follows pneumonia, relapsing fever and other acute infections, and which is probably the commonest liver disease in Africans.

The following are the most important conditions seen:—*Common infective hepatitis*, of which a small epidemic of seven cases is described. Leptospirosis, though not rigidly excluded, was unlikely, and mouse protection tests for yellow fever were negative in the one case investigated by this method. There were a few cases of *syphilitic* and *amoebic hepatitis*. *Toxic hepatitis* was diagnosed in 13 cases; neoarsphenamine accounted for 4, T.A.B. inoculations probably for 2, other chemicals for 2, and the cause was unknown in 5. No anthelmintic was incriminated, a reassuring fact. The differentiation of infective and toxic

hepatitis from yellow fever is difficult and the authors give their opinion that "every case believed to be common infective hepatitis should be investigated by mouse protection test early in the disease and again after the tenth day, to see if yellow fever immune bodies have appeared in the blood." There is a pathological note on toxic hepatitis.

Seven cases of *portal cirrhosis* and eight of *toxic cirrhosis* are described; the aetiology of the latter is that of toxic hepatitis. *Syphilitic* and *bilharzial cirrhosis* accounted each for one case. There is a pathological note on cirrhosis. *Primary hepatic carcinoma* was found in nine cases, and the authors think that toxic cirrhosis of post-degenerative type probably leads to carcinoma.

There were 19 cases of *haemolytic jaundice*; the reasons for the haemolysis were obscure, but liver injections cured the anaemia and reduced the jaundice and liver enlargement. Malaria was, apparently, not a factor in the cases reported, and obvious cases of haemolysis due to malaria, which are of course common, are not included in this survey. The anaemia in these cases of haemolytic jaundice is believed to be due to a combined dietetic deficiency of both extrinsic factor and iron.

Clinical details of these conditions are given, and the pathological and haematological findings merit attention, but cannot be abstracted satisfactorily. *C. W.*

Kolb (Lawrence C.). **Multiple Sclerosis in the American Negro.**—*Arch. Neurology & Psychiatry.* 1942. Mar. Vol. 47. No. 3. pp. 413–421. With 1 fig. [12 refs.]

Observations in Baltimore over a period of eleven years showed that multiple sclerosis is as frequent in the negro as in the white population, the respective relative rates of incidence being 17·7 and 16·8 per 100,000. These general rates compared closely with those obtained in the smaller Eastern Health District where 25 per cent. of a total population of 108,000 are negroes. The disease was seen with equal frequency in negroes of either sex.

During the period reviewed, uniform diagnostic procedures were used and before a diagnosis of multiple sclerosis was accepted a predominant number of the following criteria was required:—onset or symptoms between the ages of 15 and 55, a history of remissions and exacerbations of the disease, subjective and objective sensory disturbances, objective evidence of disease of the pyramidal tract or cerebellum, nystagmus, slurred speech, loss of abdominal reflexes, retrobulbar neuritis, presence of central scotoma, pallor of the optic discs and changes in the cerebrospinal fluid. The diagnosis was rejected if the case failed to fulfil these requirements or if another diagnosis was established through subsequent observations. All cases having a positive Wassermann reaction were also excluded although, owing to the high incidence of syphilis in American negroes, it is not unlikely that occasionally the two diseases may have co-existed.

It is suggested that the statement that multiple sclerosis is uncommon among negroes is merely an expression of opinion or rests upon analyses of inadequate data. It is impossible to state certainly whether the character and frequency of the disease in the American negro differ from those in negroes in their natural habitat. There are no adequate comparative studies from Africa and a careful review of large native populations abroad would appear desirable.

F. Murgatroyd.

BÜNGELER (W.). Ueber die Verbreitung des Rheumatismus in den tropischen und subtropischen Ländern. [**The Distribution of Rheumatism in the Tropics and Sub-tropics.**]—*Deut. Med. Woch.* 1942. Mar. 13. Vol. 68. No. 11. pp. 268–271.

This paper describes the frequency with which the author found evidence of rheumatic conditions during his work in Brazil. It was apparently inspired by a paper by VON BERGMANN in the same journal, claiming that a moderate temperature was part of the essential causative agent of rheumatic conditions, which therefore only very rarely occurred in tropical and sub-tropical regions. von Bergmann quoted American authorities to lend weight to his observations and suggested that climatotherapy should play a part in the treatment of rheumatism, and that it should be established for the treatment of such conditions in Greater Germany. The author takes exception to this suggestion and to the statements of the American writers, with which he confesses he is unfamiliar. He points out that no use of tropical or subtropical temperatures should be made in the treatment of rheumatism without an investigation into the incidence and pathology of rheumatism in the tropics. He claims here to provide the results of such an investigation.

He describes the various terrains of the State of São Paulo, where he worked, and shows how they differ in temperature, climate and humidity. He was able to perform 601 post mortem examinations, excluding those on children under 10 and lepers. Of these the majority were on poor coloured folk as it was difficult to obtain permission for autopsies from the higher social strata. The greater number of cases (403 in all) were from the city of São Paulo, others came from the interior, the coast and the dry northern uplands. In 54 of the 601 subjects (9 per cent.) he found definite evidence of rheumatic conditions. The percentages in the different districts did not vary to any great extent. Later he made a further series of examinations aimed more especially at the discovery of rheumatic conditions and claims that in 207 examinations 29 showed rheumatic conditions.

He therefore concludes that von Bergmann's findings are wrong and that the value of climatotherapy for rheumatic conditions is doubtful.

The impression given by this paper is that the author's object was more to refute von Bergmann than to produce any concrete evidence on rheumatic problems. To those accustomed to English and American ideas on the aetiology of rheumatic conditions it is strange to find rheumatism of the heart and joints always accepted as part and parcel of the same pathological process. It is also difficult to accept the author's categorical statement that all his work in Brazil goes to add further proof to the theory that the alpha and omega of the cause and treatment of rheumatism is the presence of a focus of infection, its discovery and its eradication. *W. Tegner.*

SAUNDERS (G. M.) & BANCROFT (Huldah). **Blood Pressure Studies on Negro and White Men and Women living in the Virgin Islands of the United States.**—*Amer. Heart Jl.* 1942. Mar. Vol. 23. No. 3. pp. 410–423. With 3 figs. [15 refs.]

Blood pressure records were made of 4,913 inhabitants of the Virgin Islands, representing 50 per cent. of the population over 15 years of age and made up of 4,374 negroes and 539 whites. The readings were made with a standard mercury sphygmomanometer with the patients either sitting or reclining and the appearance of the first

sound was taken as indicating the systolic pressure and the change in the sound as the diastolic pressure.

The mean systolic level for male negroes rose steadily from 120 mm. between the ages of 15 and 19, to 164 mm. at 65 to 69 years of age, after which there was a decrease. The mean systolic pressures in female negroes were essentially the same as those of males up to the age of 35 years, after which there was a more rapid increase than in the males until a peak of 184 mm. was reached at 70 to 75 years of age, after which a decrease occurred.

The systolic pressure of the white persons likewise increased with age. Statistical analysis showed that the increase in mean systolic pressures associated with advancing age was not due to increasing weight. Standardized for age, the pressure for both sexes among the negroes was considerably higher than among the whites; the male negroes had a mean systolic pressure some 12 mm. higher than male whites and the female negroes a similar increase over the female whites.

Yaws does not occur in the Virgin Islands, but as 16 per cent. of the population, representing 18 per cent. of the negroes and 4 per cent. of the whites, suffer from syphilis, a comparison was made of pressures in negroes who had positive serological reactions with the pressures in negroes who had negative reactions. The mean systolic pressures for males and females with syphilis were appreciably higher than those of persons without syphilis, but when the pressures were standardized for age there was no significant difference, because the syphilitic subjects represented an older group in which higher blood pressure was to be expected.

The diastolic pressures of female negroes were significantly higher than those of corresponding male negroes, while the pressures in whites, which were significantly lower than those of negroes, showed little difference between males and females. In all groups there was an appreciable increase with advancing age, but the rate of increase of the mean diastolic pressure was less than that for mean systolic pressure.

In the Virgin Islands the negroes have much higher, and the whites slightly higher, "normal" blood pressures than published standards for North America, and, compared with residents of the U.S.A., a greater percentage of the negroes have systolic pressures above 150 mm. or below 110 mm. The excess of low blood pressure may be a result of climate and general environment, as it is fairly generally agreed that blood pressure is lower in a tropical environment than in colder localities. As the inhabitants of the Virgin Islands are not subject to the stress and hurry of modern civilization some other explanation must be invoked for the higher pressures, and as the islands are poor and do not yield agricultural products in any quantity, it is suggested that poverty, with its associated vitamin deficiencies, may explain the tendency to raised blood pressure. *F. Murgatroyd.*

DE WAAL (H. L.). **South African Senecio Alkaloids. Part 5. Notes on Isatidine, Rosmarinine and Pterophine, and on the Structure of their Necines and Necic Acids.**—*Onderstepoort Jl. Vet. Sci. & Animal Industry.* 1941. Jan. & Apr. Vol. 16. Nos. 1 & 2. pp. 149–166. With 3 figs. [18 refs.]

This article deals entirely with the chemical aspect of the subject which, though important, will not appeal to readers of this *Bulletin.* *H. Harold Scott.*

Steyn (Douw G.) & van der Walt (S. J.). **Recent Investigations into the Toxicity of Known and Unknown Poisonous Plants in the Union of South Africa, XI.**—*Onderstepoort Jl. Vet. Sci. & Animal Industry.* 1941. Jan. & Apr. Vol. 16. Nos. 1 & 2. pp. 121–147. With 7 figs. [18 refs.]

The authors have carried out investigations and experimental work on 16 species of plants in South Africa. Three were definitely proved to be poisonous, *viz.*, *Senecio pterophorus*, *Kalanchoe paniculata* and *K. thyrsiflorae*. Isatidine and retrorsine, alkaloids of Senecio, produced all the symptoms of, and the same histological changes observed in, a disease of horses known as *dunsiekte*. Two other species, *Crotolaria rhodesiae* and *Pseudogaltónia clavata* may be, but have not yet been proved to be, poisonous. All the work referred to in this article has been carried out on animals. [See *Bull. of Hyg.*, 1936, Vol. 11, p. 849; 1937, Vol. 12, p. 898; 1941, Vol. 16, p. 137; and this *Bulletin*, 1941, Vol. 38, p. 421.] *H. Harold Scott.*

Roy (D. N.) & Ghosh (S. M.). **A New Active Constituent of Pyrethrum Flower.** [Correspondence.]—*Nature.* 1942. Aug. 1. Vol. 150. No. 3796. p. 153. With 1 chart.

It has usually been held that the insecticidal property of extracts of pyrethrum is due to the pyrethrins, but the authors have found, in a series of tests with *Musca vicina*, that there is no direct relation between the proportion of insect kills and the amount of pyrethrin in the extracts, and that a smaller amount of pyrethrin extracted from a sample of pyrethrum with a high pyrethrin value produces better results than a larger amount from a poorer sample. They therefore assume that the lethal principle is not pyrethrin, but is probably a substance which exists in larger amounts in samples rich in pyrethrin; it is probably an independent body whose growth in the flowers is very likely regulated by the same factors as pyrethrin. It is soluble in kerosene (which was used in the experiments but which is not itself lethal). *C. W.*

Anderson (W. M. E.). **Anti-Sandfly Spraying with Lethane and Pyrethrum.**—*Jl. Roy. Army Med. Corps.* 1942. July. Vol. 79. No. 1. pp. 12–24. With 2 figs.

The work was carried out in Peshawar, N.W. India, rooms being sprayed with certain dilutions of Lethane 384 and with various extracts of pyrethrum. Sandflies were numerous, the majority of them *Phlebotomus papatasii*. The spraying was done with small hand machines, and also with Phantomysts which fill the room with a fine mist of insecticide. As a result of his work the author recommends the use of either Lethane 384, diluted 1/66, or "1/100 Pyefly." The actual amount of Lethane to be used is not stated, but the reader can calculate it from the body of the paper; we have not found it possible to calculate the final concentration in pyrethrins or the quantity to be used per unit of space. The author refers to the "repellent effect" of the insecticides. His tables show that certain doses of either of these insecticides reduce the number of sandflies for 12 hours, or occasionally a longer period. This either shows that the insecticide only kills a small proportion of the insects, or that others move into the room after treatment, but it is not evidence that the materials are repellent.

P. A. Buxton.

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Am. signifies Amoebiasis and Amoebic Dysentery.
Bb. ,, Beriberi
Bl. ,, Blackwater.
B.R. ,, Book Review.
Chl. ,, Cholera.
Der. ,, Tropical Dermatology.
Diet. ,, Deficiency Diseases including Epidemic Dropsy.
Dys. ,, Dysentery (Bacillary and Unclassed).
Fev. ,, Fevers.
Haem. ,, Haematology.
Hel. ,, Helminthiasis.
Leish. ,, Leishmaniasis.
Lep. ,, Leprosy.
Lept. signifies Leptospirosis.
Mal. ,, Malaria.
Misc. ,, Miscellaneous.
Oph. ,, Tropical Ophthalmology.
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Pl. ,, Plague.
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R.B.F. ,, Rat-Bite Fever.
R.F. ,, Relapsing Fever and other Spirochaetoses
Sp. ,, Sprue.
Spec. Res. signifies Special Research.
Tryp. signifies Trypanosomiasis.
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Corwin, W. C., 322 (Dys.)
Cosgrove, P. C., with Cole & Robinson, 746 (Leish.)
Costa, O. & Junqueira, M. A., 107 (Der.)
Costa, R. S., with Talice & Marra, 536 (Tryp.)
Cot Lesmes, V., 219 (Lep.)
Couch, J. F., with Briese, 419 (Misc.)
Coulston, F. & Manwell, R. D., 440 (Mal.)

Coulston, F., with Mueller, 89 (Hel.)
Coutinho, J. O., 610 (Leish.), 738 (Mal.)
—— & Barreto, M. P., (611) (Leish).
——, with Galvão, (611) (Leish.)
——, with Pessôa, 175, 610 (Leish.)
Covell, G., 205 (B.R.), 259 (Mal.)
Cowdry, E. V. & Ruangsiri, C., 233 (Lep.)
Cox, H. R., 381 (Typh.)
Craig, C. F. & Faust, E. C., 58 (B.R.)
Cram, E. B., 191 (Hel.)
——, Jones, M. F. & Reardon, L., 191 (Hel.)
Creel, R. H., 303 (Pl.)
Crocket, J. A., with Murray, 580 (Spec. Res.)
Cross, J. C., 653 (Misc.)
Crowell, R. L., with Smith & Watson, 392 (Mal.)
Cruickshank, R., with Anderson, 34 (Dys.)
Cruz, J. R., with Vitug & Bautista, 11 (Hel.)
Cuéllar, P. I. V., 38 (Y. & S.)
Cuénod, A., with Burnet & Nataf, 888 (Oph.)
Culbertson, J. T., 32 (Am.), 355 (B.R.)
—— & Greenfield, S. H., 631 (Hel.)
—— & Kessler, W. R., 607 (Tryp.)
—— & Rose, H. M., 95 (Hel.)
Cutting, W. C., with Kuzell, Layton & Frick, 318 (Am.).

D.

Daengsvang, S. & Mangalasmaya, M., 564 (Hel.)
Daigh, F. C., with Mentzer & Connell, 491 (Misc.)
Daleas, P., 57 (Misc.), 179 (Mal.)
Dameshek, W., 639 (Haem.)
Dammin, G. J., 194 (Hel.)
—— & Billings, F. T., Jr., 822 (Typh.)
Damon, S. R. & Sellers, T. F., 590 (Rab.)
Dangerfield, L. F. & Gear, J., 108 (Der.)
Dang-van-Ngu, with Galliard, 13 (Hel.)
Dao Van Ty, with Ristorcelli, (833) (Fev.)
Darman, M., 745 (Tryp.)
Das, B. C., with Greval & Chandra, 94 *bis*, (Hel.), 461 (Lep.)
——, with —— & Sen Gupta, 225 (Lep.)
Das, P. C., with Read & Pandit, 838 (Chl.)
Das, S., with Viswanathan, Oommen, 598 (Mal.)
Das Gupta, A. C., with Lal, 713 (Sp.)
——, with Lal, Agarwala & Adak, 340 (Diet.)
——, with ——, Mukherji & Adak, 340 (Diet.)
Das Gupta, B. M., 347 (Lept.)
Das Gupta, C. R., 201 (Misc.)
——, with Napier & Majumdar, 100 (Hel.)
——, with —— & Neal-Edwards, 481 (Haem.)
Daubney, R. & Hudson, J. R., 533 (Tryp.)
Dauncey, K. E. D., 256 (Mal.)
Davey, D. G. & Innes, J. R. M., 861 (Hel.)
Davey, T. F., 695, 850 (Lep.)
Davey, T. H., with Gordon & Peregrine, 515 (Mal.)
Davidson, W. B., 201 (Misc.)
Davies, J. N. P. & Hewer, T. F., 487 (Misc.)
Davis, C. L., with Stiles, 790 (Der.)
Davis, D. H. S., with Gear, 750 (Typh.)
Davis, G. E., 125, 343, 344 (R.F.)
——, Wynns, H. L. & Beck, M. D., 556 (R.F.)
Davis, W. T., 889 (Oph.)
Davison, A. R. & Grasset, E., 232 (Lep.)
Davson, J., 568 (Hel.)
Dawson, J. R., Jr., 80 (Rab.)
Dayal, S., 889 (Oph.)
Dayalu, T. K. & Jayaram, S. S., 317 (Am.)
De, S. S., with Ghosh & Chowdhury, 405, 718 (Vms.)
DeBakey, M., with Ochsner, 840 (Am.)
DeEds, F. & Thomas, J. O., 6 (Hel.)
Deeny, J., 335 (Pel.)
De la Barrera, J. M., 305 (Pl.)
Delaunay, A., with Boquet, 884 (Vms.)
Della Vida, B. L. & Dyke, S. C., 197 (Hel.)
Del Ponte, E., (157) (Fev.)
DeMonte, A. J. H., with Chopra & Chatterji, 459 (Chl.)
—— & Gupta, S. K., 164 (Chl.)
——, with Pasricha & Lahiri, 162 (Chl.)
Denecke, K., 348 (Misc.)
Derry, D. C. L., Card, W. I. & Wilson, R., 645 (Der.)
Deschiens, R. & Nicol, L., (102) (Hel.)
—— & Renaudet, R., (93) (Hel.)
Des Ligneris, M. J. A., 578 (Misc.)
Desportes, C., with Brumpt, 829 (Typh.)
Deucher, W. G., with Hotz, 44 (Sp.)
Devine, J. & Fulton, J. D., 438 (Mal.)
Dey, N. C. & Maplestone, P. A., 103 (Der.)
Dharmendra, 200 (Misc.), 220, 227, 228, 855 (Lep.)
—— & Jaikaria, S. S., 226 (Lep.)
—— & Lowe, J., 696, 856, 857 (Lep.)
——, with ——, 227 (Lep.)
——, with —— & Sen, 557 (Lep.)
Dhayagude, R. G., 413 (Misc.)
—— & Amin, B. M., 707 (Hel.)
Dickson, W. E. C. & Willis, J. D., 92 (Hel.)
Dietrich, A., (755) (Typh.)
Diggs, L. W. & Pettit, V. D., 716 (Haem.)
Dikshit, B. B., 524 (Mal.)
Dillenberg, H., with Eyer & Przybylkiewicz, 385 (Typh.)
Diño, B. R., de Leon, W. & Galvez, A. A., 466 (Hel.)
Do Amaral, A. D. F., 175 (Leish.)
Dobler, M., with Hegner & West, 398 (Mal.)
——, with ——, —— & Ray, 124 (Mal.)
Dodds, G. E., 889 (Oph.)
Dodero, J., 82, 87 (Rab.)
——, with Auffret, 82 *bis* (Rab.)
—— & Hoang-Tich-Try, 82 (Rab.)
Donaldson, L., with Humphreys, 240 (Tryp.)
Donelson, E. G., with Leichsenring & Wall, 480 (Haem.)
Donohue, W. L., with Brick, 571 (Hel.)
Donovan, A. & Hopkins, E. D., 312 (Pl.)
Dormanns, E. & Emminger, E., 824 (Typh.)
Dormer, B. A. & Friedlander, J., 31 (Am.)
Dörschel, with Jacobi, 754 (Typh.)
Dostrovsky, A. & Sagher, F., 749 (Leish.)
Douglas, J. R., with Wheeler, 616 (Pl.)
——, with —— & Evans, 307 (Pl.)
Doull, J. A., Rivera, E. M., Saunders, G. M., Guinto, R. S. & Morales, E. G., 219 (Lep.)
Dove, W. S., 804 (Mal.)
Dowdeswell, R. M., (688), 836 (Pl.)
——, with Sequeira, 796 (Misc.)
Drechsler, C., (493) (Misc.)
Drensky, K., 602 (Mal.)

Du Bois, 102 (Der.)
Dubois (A.), (857) (Lep.)
——, with Dupont, 849 (Y. & S.)
—— & Gavrilof, W., 621 (Lep.)
Duckworth, G., 785 (Diet.)
Duff, D., 76 (Y.F.)
Dulaney, A. D., Stratman-Thomas, W. K. & Warr, O. S., 119 (Mal.)
Dunn, M., with Boyd, 439 (Mal.)
Dupont, A. & Dubois, A., 849 (Y. & S.)
Durand, P., 589 (Rab.)
—— & Balozet, L., 369, 543 (Typh.)
—— & Giroud, P., 546 (Typh.)
——, —— & Sparrow, H., 546 (Typh.)
Durieux, C., with Peltier, Jonchère & Arquié, 761 (Y.F.)
Du Toit, M. L., 809 (Mal.)
Dutta, U. C., with Woodhead, 147 (Typh.)
Dwork, K. G., 749 (Leish.)
Dy, F. J., with Africa, (438) (Mal.)
——, with —— & Soriano, 27 (Mal.)
Dyer, B. R., 410 (Misc.)
Dyer, R. E., 453 (Typh.)
van Dyke, H. B., with Walker, 441 (Mal.)
Dyke, S. C., with Della Vida, 197 (Hel.)

E.

Eagle, H., Mays, J. R. S., Hogan, R. B. & Burney, L. E., 119 (Mal.)
Earle, K. V., 49, (351), 486, 792 (Misc.), 331 (Bb.), (327), 328 (Diet.), 571 *bis*, 572 (Hel.)
Earle, W. C., with Vargas & Casis, 430 (Mal.)
East African Medical Journal, 793 (Misc.)
Ebenezer, R., with Sloan, 559 (Lep.)
Edwards, L. B., 320 (Dys.)
Edwards, M. I. N., with Napier, 785 (Haem.)
Efremov, V. V., 478 (Diet.)
Egan, E., 463 (Hel.)
Egorova, O. G., with Sablin, 519 (Mal.)
Ejercito, A., 24, (438), 523 (Mal.)
—— & Celis, E. B., 262 (Mal.)
Ellerman, J. R., 205 (B.R.)
Elliott, M., 778 (Hel.)
Emerson, G. A., 230 (Lep.)
Emminger, E., with Dormanns, 824 (Typh.)
Emmons, C. W., Hailey, Howard & Hailey, Hugh,, 106 (Der.)
Engineering News-Record, 664 (Mal.)
Errington, B. J., 6 (Hel.)
Escalona, E., (225) (Lep.)
Escobar, J. J., 113, 643 (Der.)
Eskola, O., 864 (Hel.)
Espin, J., 466, 628 (Hel.)
Estibotte, R. & Roussel, H., 888 (Oph.)
Estrada, J. & Garcia, E., 101 (Hel.)
Essick, H. C., with Tucker & Woodring, 145 (Typh.)
Evans, F. C., with Stewart, 616 (Pl.)
——, with Wheeler & Douglas, 307 (Pl.)
Ewing, C. W., 92 (Hel.)
Eyer, H., Przybylkiewicz, Z. & Dillenberg, H., 385 (Typh.)

F.

Faget, G. H., 695, 852 (Lep.)
—— & Johansen, F. A., 698 (Lep.)
Fahr, T., 528 (Bl.)
Faiguenbaum, J., with Neghme, 246 (Tryp.)
Fair, G. M., with Chang, 313 (Am.)
Fairley, N. H., 270, 271 (Bl.)
Farrell, E., (278) (Misc.)
Fasal, P., 327, (328) (Diet.)
Fauconnier, H., with Anderson, Berge & Runacher, 342 (R.F.)
Fauconnier, J., with Berge, Audoyé & Berre, 703 (Hel.)
Faust, E. C., 350 (Misc.), 388 (Mal.), 554 (Am.)
——, with Craig, 58 (B.R.)
——, with Sawitz, 765 (Am.)
——, Thomas, E. P. & Jones, J., 189 (Hel.)
Federated Malay States, 258 (Mal.)
Fehily, L., 332, (334) *bis* (Bb.)
Feldt, A., 344 (R.F.)
Fellows, F. S. & Perry, W. B., 387 (Mal.)
Feng, L.-C., with Chung, 169 (Leish.)
Fenyuk, B. K. & Sheikina, M. V., 308 (Pl.)
Fernandez, J. M. M. & Olmos Castro, N., 227 (Lep.)
Fernando Videla, L., with de Villafañe Lastra & Sosa Gallardo, 309 (Pl.)
Ferracani, R. S., 706 (Hel.)
Ferrari, A., 293 (Mal.)
Ferrer, I., with Pardo-Castello, 789 (Der.)
Field, J. W., 17, 254 (Mal.)
——, Niven, J. C. & Mitchell, J. F., 179, 182 (Mal.)
de Figueiredo, F. V., 43 (Sp.)
Figuerero, M. L., with Toranzos & Barvara, 244 (Tryp.)
Figueroa, I., 433 (Mal.)
Findlay, G. M., (65), 65 *bis*, 68, (79), 761 (Y.F.), 367, 380, 383, 682 (Typh.)
——, Kirk, R. & Lewis, D. J., 457 (Y.F.)
——, —— & MacCallum, F. O., 455 (Y.F.)
Fischer, A., 636 (Diet.)
Fischer, O. & Staupendahl, W., 434 (Mal.)
Fisher, A. C., 199 (Misc.)
Fisher, R. C., with Vance, 716 (Haem.)
Fite, G. L., 221, 223 (Lep.)
Fitzpatrick, F. & Hampil, B., 674 (Typh.)
Flegontovo, A. A., with Rall' & Sheikina, 307 (Pl.)
Flippin, H. F., with Schwartz & Furst, 601 (Mal.)
Fonte, J., 224 *bis* (Lep.)
Fosdick, R. B., 64, 615 (Y.F.)
Fox, C. L., 272 (Bl.)
Fox, L. A., with Rozeboom & Laird, 430 (Mal.)
Foy, H., Kondi, A. & Moumjidis, A., 266 (Bl.)
—— & Lewis, E. G., 268 (Bl.)
Fraenkel, E. M., 375, (680) (Typh.)
Franseen, C. C., 47 (Misc.)
Fraser, P. K., 103 (Der.)
Free, A. H., with Beams & Glenn, 334 (Pel.)
Freire, R. S., with Mazza & Salica, 671 (Tryp.)
Frick, W. D., with Kuzell, Layton & Cutting, 318 (Am.)
Fricks, L. D., 521 (Mal.)

Friedheim, E. A. H., 444, 819 (Tryp.)
Friedlander, J., with Dormer, 31 (Am.)
Frontali, G., 709 (Diet.)
Frota, P., with de Paula, 315 (Am.)
Fulton, J. D., 815 (Mal.)
——, with Devine, 438 (Mal.)
—— & Yorke, W., 438 (Mal.), 533 (Tryp.)
Furst, W., with Schwartz & Flippin, 601 (Mal.)

G.

Gabaldon, A., with Rozeboom, (431) (Mal.)
Gajardo Tobar, R., 641, 642((Vms.)
Gale, G. W., 302 (Pl.)
Galliard, H. & Dang-van-Ngu, 13 (Hel.)
—— & v. Ngu, D., (16) (Hel.)
Gallo, P. & Iturbe, J., 87 (Rab.)
Galvão, A. L. A. & Coutinho, J. O., (611) (Leish.)
Galvez, A. A., with Dino & de Leon, 466 (Hel.)
Garcia, E., with Estrada, 101 (Hel.)
Garcia, E. Y., 116 (Mal.)
——, with Africa, 467 (Hel.)
Garcia, J. C., 96 (Hel.)
Garcia Caballero, A. & Mayoral, P., 682 (Fev.)
García Capurro, F., 569 (Hel.)
Garcia Palazuelos, P., with Alessandri & Lerner, 710 (Diet.)
—— & Lerner, J., (556) (Am.)
Gasic, G. & Bertín, V., 246 (Tryp.)
Gast Galvis, A., 73 (Y.F.)
Gault, E. S., with Henderson & Spaulding, 767 (Lep.)
Gaupp, H., (706) (Hel.)
Gauran, M., with Sédan, Koutseff & Livon, 888 (Oph.)
Gautier, R., 84 (Rab.)
Gavrilof, W., with Dubois, 621 (Lep.)
Gear, J., (48) (Misc.)
——, with Dangerfield, 108 (Der.)
—— & Davis, D. H. S., 750 (Typh.)
——, Harris, L. C. & Saner, R. G., 830 (Typh.)
—— & de Meillon, B., 152 (Typh.)
——, with van Zyl, 236 (Tryp.)
Geldrich, J., 673 (Leish.)
Gelfand, M., 10, 699 (Hel.)
Genevray, J. & Try, H. T., 177 (Mal.)
Ghosh, B. N., with Chaudhuri, 719 (Vms.)
——, De, S. S. & Chaudhuri, D. K., 405 (Vms.)
——, —— & Chowdhury, D. K., 718 (Vms.)
Ghosh, H. & Mukerjee, S., 690 (Chl.)
Ghosh, J. & Roy, B., 341 (Diet.)
Ghosh, R. K., 278 (Misc.)
Ghosh, S. K., with Panja, Malik & Paul, 839 (Chl.)
Ghosh, S. M. with Chaudhury, 833 (Fev.)
——, with Chopra & Roy, 260 (Mal.)
——, with Roy, 900 (Misc.)
——, with —— & Chopra, 795 (Misc.)
Giampietro, J., with Saldun de Rodriguez & Soto, 704 (Hel.)
Gibbins, E. G., 442 (Tryp.) (632) (Hel.)
Gildemeister, E. & Haagen, E., 681 (Typh.)
Gill, G. A., 65 (Y.F.)
Gillett, J. D., 650 (Misc.)
——, with Mahaffy, Smithburn & Jacobs, 759 (Y.F.)
Gilligan, D. R., with Altschule, 274 (Bl.)
Gilmore, H. R., Jr., Kean, B. H. & Posey, F. M., Jr., 796 (Misc.)
Girard, G., 308, 763 (Pl.)
Giri, K. V., (574) (Diet.)
—— & Naganna, B., (336) (Pel.)
Giroud, P., with Durand, 546 (Typh.)
——, with —— & Sparrow, 546 (Typh.)
—— & Panthier, R., 753 (Typh.)
Gispen, R., 147 (Typh.)
Githens, T., S., 407 (Vms.)
Gleitsmann, H. W., 893 (Misc.)
Glenn, P. M., with Beams & Free, 334 (Pel.)
Góbich, E., with Rivas, (706) (Hel.)
Gohar, M. A., 161 (Chl.)
Goldberger, J., with Sotolongo, 647 (Misc.)
Golden, R., with Lepore, 337 (Pel.)
Goldstein, G., 689 (Pl.)
Gomes, B. F., with Versiani, 400 (Mal.)
Gomes, J. M., 559 (Lep.)
Gomes, L. de S. & Jordão, S., 890 (Oph.)
Gomez, F. B., with Gutierrez, Navarro, Morales & Lozano, 460 (Y. & S.)
Gomez Farias, R., 484 (Der.)
Gómez Plata, C., 558 (Lep.)
Gonzalo Guerra, (695) (Y. & S.)
Gordon, E. F., 252 (Mal.)
Gordon, J. & Johnstone, K. I., 617 (Chl.)
Gordon, R. M., 54 (Misc.)
——, Davey, T. H. & Peregrine, E. P., 515 (Mal.)
Grachev, Y. S., 517 (Mal.)
Gradle, H. S., 888 (Oph.)
Grana, A. & Schenone, B. H., 705 (Hel.)
Grasset, E., 311, 312 (Pl.), 407 (Vms.)
——, with Davison, 232 (Lep.)
——, with Pirie, 311 (Pl.)
Green, B. H., with Block & Tarnowski, 36 (Dys.)
Greenfield, J. G., with Hawking, 235 (Tryp.)
Greenfield, S. H., with Culbertson, 631 (Hel.)
Greengard, J. & Hess, E. R., 127 (R.B.F.)
Greval, S. D. S., 461 (Lep.)
——, Chandra, S. N. & Das, B. C., 94 *bis* (Hel.), 461 (Lep.)
——, Das, B. C. & Sen Gupta, P. C., 225 (Lep.)
Griffitts, J. J., 764 *bis* (Chl.)
Gromashewskij, L. W., 141 (Typh.)
Groot, H., 452 (Typh.)
——, Mayoral, P. & Martínez, L. E., 140, 678 (Typh.), (548), 683 *bis* (Fev.)
Guerra, G., 41 (Y. & S.).
Guillerm, J., with Chaussinand, 230 (Lep.)
Guimarães, R., with de Magalhães, 409 (Vms.)
Guindy, 161 (Chl.)
Guinto, R., with Doull, Rivera, Saunders & Morales, 219 (Lep.)
Guinto, R. S. & Rodriguez, J. N., 216, 217 (Lep.)
Gunewardene, S. R., with de Saram, 570 (Hel.)
Gupta, B. P., with Bhattacharya, 690 (Chl.)
Gupta, J. C., with Chopra & Sen, 255 (Mal.)
Gupta, P. C. S., with Napier, 748 (Leish.)
Gupta, S. K., with deMonte, 164 (Chl.)
Gutierrez, P. D., Navarro, R. J., Morales, S. B., Lozano, A. A. & Gomez, F. B., 460 (Y. & S.)

H.

Haagen, E., with Gildemeister, 681 (Typh.)
Habel, K., 81 (Rab.)
Haddow, A. J., 595 (Mal.)
——, with Symes & McMahon, 577 (Misc.)
Haga, J., (620) (Am.)
Hahn, P. F., with Yuile, Steinman & Clark, 276 (Bl.)
Hailey, H., with Emmons & Hailey, Hugh, 106 (Der.)
Hala, H. L., 571 (Hel.)
Halder, K. C., with Smith & Ahmed, 447, 449 (Leish.)
Hallmann, L., 678 (Typh.)
Hamburger, H. J., 896 (Misc.)
Hampil, B., with Fitzpatrick, 674 (Typh.)
Hand, R. F., with Southwell-Sander, 393 (Mal.)
Hanks, J. H., 220 (Lep.)
Hare, K. P., 418 (Misc.)
Hargett, M. V., 458 (Y.F.)
——, with Berge, 686 (Y.F.)
Hargreaves, W. H. & Mackenzie, K. G. F., 409 (Vms.)
Harley, G. W., (493) (Misc.), 724 (B.R.)
Harris, L. C., with Gear & Saner, 830 (Typh.)
Harris, S. & Harris, S., Jr., 637 (Sp.)
Harris, S., Jr., with Harris, 637 (Sp.)
Hartman, H. R. & Kyser, F. A., 32 (Am.)
——, —— & Comfort, M. W., 621 (Am.)
Hawking, F., 125 (R.F.), 534 (Tryp.)
—— & Greenfield, J. G., 235 (Tryp.)
—— & Smiles, J., 238 (Tryp.)
Hawkins, J. W., with Aycock, 219 (Lep.)
Hayman, R. W. & Holt, G. W. C., 205 (B.R.)
Hayward, J. C., with Kark & Souter, 45 (Sp.)
Hearin, J. T., 93 (Hel.)
Hegner, R., West, E., Ray, M. & Dobler, M., 124 (Mal.)
——, —— & Dobler, M., 398 (Mal.)
Heilig, R., 569 (Hel.)
—— & Naidu, V. R., 375, 827 (Typh.)
Henderson, H. J., 766 (Lep.)
Henderson, R. G., Pinkerton, H. & Moore, L. T., 645 (Der.)
Hennessey, R. S. F., with Mowat, 413 (Misc.)
——, with Muwazi & Trowell, 896 (Misc.)
Henriques, A., 551 (Pl.)
Henry, A. F. X., (256) (Mal.)
Hernandez Morales, F., with Carrión & Ruiz Nazario, 110 (Der.)
Hernando Ordoñez, J., 648 (Misc.)
Hertig, M., 831 (Fev.)
Hess, A. D. & Tarzwell, C. M., 604 (Mal.)
Hess, E. R., with Greengard, 127 (R.B.F.)
Hetsch, H., 548 (Typh.)
Hewer, T. F., 65 (Y.F.)
——, with Davies, 487 (Misc.)
Hewitt, R., with Hurlbut, 295 (Mal.)
—— & Kotcher, E., 261 (Mal.)
Hicks, H. M., with Hoff, 792 (Misc.)
Hill, T. R., 803 (Mal.)
Hillman, R. W., 877 (Hel.)
Ho, E. A., Chu, H. J. & Yuan, I. C., 169 (Leish.)
Hoang-Tich-Try, with Dodero, 82 (Rab.)
Hoch, P., Kusch, E. & Coggeshall, L. T., 20 (Mal.)
Hodgkin, E. P., with Tweedie & Reid, 522 (Mal.)
Hoff, A. & Hicks, H. M., 792 (Misc.)
Hoffman, W. A., 350 (Misc.)
—— & Janer, J. L., 561 (Hel.)
——, with Krakower & Axtmayer, 562 (Hel.)
Hogan, R. B., with Eagle, Mays & Burney, 119 (Mal.)
Holinger, P. H., with van Pernis & Benson, 109 (Der.)
Holder, H. J., with McKelvie & Kirk, 401 (Oph.)
Hollack, J., with v. Bormann & Kirchhöfer, 894 (Misc.)
Holland, G. P., 306 (Pl.)
Holm, B., 670 (Bl.)
Holt, G. W. C., with Hayman, 205 (B.R.)
van Hoof, L., 38 (Y. & S.), 815 (Tryp.)
Hopkins, E. D., with Donovan, 312 (Pl.)
Hopkins, G. H. E., 391 (Mal.)
Hoque, A. K. M., (555) (Am.)
Hornby, H. E., 242 (Tryp.)
Hoskins, W. M., 490 (Misc.)
Hotz, H. W. & Deucher, W. G., 44 (Sp.)
Howe, C., 386 (Fev.)
Hoyne, A. L. & Larimore, G. W., 486 (Misc.)
Hrad, O., 609 (Leish.)
Hu, S. M. K., 185, 708 (Hel.)
Huaman, N., with Mendez, 432 (Mal.)
Huang, M. S., with Kuo, 338 (Pel.)
Huart, 127 (R.B.F.)
Hubble, D., 464 (Hel.)
Hudson, J. R., with Daubney, 533 (Tryp.)
Hudson, N. P., 153 (Typh.)
——, with Riddle & Spies, 339 (Pel.)
Hueck, O., 893 (Misc.)
Huff, C. G., 124 (Mal.), (351) (Misc.)
Hughes, T. P., Jacobs, H. R. & Burke, A. W., 455 (Y.F.)
——, with Mahaffy, Smithburn & Kirk, 456 (Y.F.)
——, with Lewis & Mahaffy, 685 (Y.F.)
Hughes, W., 802 (Mal.)
—— & Baldwin, R. B. T., 756 (Typh.)
Hühne, W., 663 (Mal.)
Humphreys, D. R., 864 (Hel.)
Humphreys, E. M. & Donaldson, L., 240 (Tryp.)
Humphreys, R. M., 749 (Leish.)
Hundertmark, A., with Weyer, 428 (Mal.)
Hurlbut, H. S., (295) (Mal.)
—— & Hewitt, R., 295 (Mal.)
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Nataf, R., with Burnet & Cuénod, 888 (Oph.)
Nauck, E. G., 367 (Typh.), 833 (Y.F.)
—— & Weyer, F., 366, 383 (Typh.)
—— & Zumpt, F., 366 (Typh.)

Navajas, E., with Cardosa, 248 (Tryp.)
Navarro, R. J., with Gutierrez, Morales, Lozano & Gomez, 460 (Y. & S.)
Neal-Edwards, M. I., with Napier & Das Gupta, 481 (Haem.)
Neghme, A. & Faiguenbaum, J., 246 (Tryp.)
——, with Noé, 427 (Mal.)
de Negri, U., 262, 520 (Mal.)
Neitz, W. O., Alexander, R. A. & Mason, J. H., 757 (Typh.)
Neiva, C. & Arantes, J. B., 406 *ter* (Vms.)
——, with ——, 405, 641 *bis* (Vms.)
Nettel, R., 572 (Hel.)
v. Ngu, D., with Galliard, (16) (Hel.)
Nicholls, L., 338 (Pel.)
Nicol, L., with Deschiens, (102) (Hel.)
——, with Ramon, Boquet & Richou, 886 (Vms.)
Nicol, W. D. & Shute, P. G., 739 (Mal.)
Nigeria, 442 (Tryp.)
Niño, F. L., 107 (Der.), (350) (Misc.)
Niven, J. C., 258 (Mal.)
——, with Field & Mitchell, 179, 182 (Mal.)
Noé, J. & Neghme, A., 427 (Mal.)
Nolasco, J. O. & Lara, C. B., 222 (Lep.)
Noronha, A. J., 18 (Mal.)
Nunemaker, J. C., with Brown, 693 (R.F.)

O.

Ocaña, T., with Scolari, 565 (Hel.)
Ochsner, A. & DeBakey, M., 840 (Am.)
O'Donovan, D. K., McGrath, J. & Boland, S. J., 692 (Dys.)
Oelkers, H. A. & Rathje, W., 767 (Hel.)
—— & Zessler, H., 709 (Hel.)
Oesterlin, M., 299 (Mal.)
Oettel, H. & Thaddea, S., 638 (Sp.)
Offutt, E. P., Jr. & McCoy, O. R., 635 (Hel.)
Ohama, S., 97, 183 *bis* (Hel.)
O'Leary, S. B., with Moll, 303, 687 (Pl.)
Oliver-González, J., (627) (Hel.)
Olleros, A. R., 480 (Sp.)
Olmos Castro, N., with Fernandez, 227 (Lep.)
Omori, N., 55 (Misc.)
Oommen, A. V., with Viswanathan & Das, 598 (Mal.)
Oomen, H. A. P. C., 39 (Y. & S.)
Ordman, D., 343 (R. F.)
Orissa, 158 (Chl.), 216 (Lep.)
Orlina, M. M., 519 (Mal.)
Ortiz, A. A., 465 (Hel.)
Ortiz, J., with Stokes, 618 (Am.)
Osburn, H. S., 313, 619 (Am.)
Osorio, M. T., with Mazzotti, 704 (Hel.)
Otálora, B., 820 (Tryp.)
Otten, L., 309 (Pl.), (484), (791) (Misc.)
Otten-van Stockum, M. J., 592 (Rab.)
Otto, G. F., with Brown, 99 (Hel.)
Otto, R. & Bickhardt, R., 538 (Typh.)
Ou, T., 118 (Mal.)
Ovalle, H., with Macchiavello & Cifuentes, 374 (Typh.)
Ovares, J. C., with Peña Chavarría, 644 (Der.)
Owczarewicz, L., 751 (Typh.)

P.

Pacheco Marroquin, S., 882 (Sp.)
Paldrok, A., 462, (857) (Lep.)
Palestine, 592 (Mal.)
Palmer, E. D., 98 (Hel.)
Panama Canal, (575) (Misc.)
Pande, P. G., 537 (Leish.)
Pandit, S. R., with Read ,159 (Chl.)
——, with —— & Das, 838 (Chl.)
Pandya, B. C., Nargund, K. S. & Bokil, K. V., (698) (Lep.)
Panja, G., Malik, K. S., Paul, B. M. & Ghosh, S. K., 839 (Chl.)
Panthier, R., with Giroud, 753 (Typh.)
Para, M., with Villela, 645 (Der.)
Pardal, E., 762 (Pl.)
Pardo-Castello, V. & Ferrer, I., 789 (Der.)
Parise, N., 22 (Mal.)
—— & Lucrezi, G., 516 (Mal.)
Parker, W. V., with Knowles & Johnson, 123 (Mal.)
Parpacén, J. V., (8) (Hel.)
Pascale, H., with Pessoa, 473 *quin.*, (476) (Hel.)
Pasricha, C. L. & Abedin, Z., 407 (Vms.)
——, —— & Paul, B. M., 166 (Chl.)
——, Lahiri, M. N. & deMonte, A. J. H., 162 (Chl.)
——, Malik, K. S. & Paul, B. M., 166 (Chl.)
—— & Paul, B. M., 162 (Chl.)
Paterno, P., (254) (Mal.)
Pathan, H. A. H., 886 (Oph.)
Patiño-Camargo, L., 378, 542 (Typh.)
Patrick, D. W. & Wolfe, D. M., 226 (Lep.)
Pattanayak, G. C., 177 (Mal.)
Paul, B. M., with Panja, Malik & Ghosh, 839 (Chl.)
——, with Pasricha, 162 (Chl.)
——, with —— & Abedin, 166 (Chl.)
——, with —— & Malik, 166 (Chl.)
de Paula, H. V. & Frota, P., 315 (Am.)
Pawel, M., 574 (Diet.)
Pecache, L. V., with Stransky, 411 (Misc.)
Pechtel, C., 386 (Fev.)
Peltier, M., Durieux, C., Jonchère, H. & Arquié, E., 761 (Y.F.)
Peña Chavarría, A. & Ovares, J. C., 644 (Der.)
Peña Yañez, A., 386 (Fev.)
Penfound, W. T., 740 (Mal.)
Penner, L. R., 769 (Hel.)
Peregrine, E. P., with Gordon & Davey, 515 (Mal.)
Pereira, M. de C., with de Meillon, 294 (Mal.)
Pereira, O. de L., 698 (Lep.)
Perfetti, A., with Vaccaro, 752 (Typh.)
van Pernis, P. A., Benson, M. E. & Holinger, P. H., 109 (Der.)
Perrin, T. L. & Bengtson, I. A., 830 (Typh.)
Perry, W. B., with Fellows, 387 (Mal.)
Pesce, H., (219), 623 (Lep.)
Pessin, S. B., with Stovall, 590 (Rab.)
Pessôa, S. B., 175, 673 (Leish.)
—— & Coutinho, J. O., 175, 610 (Leish.)
—— & Lucena, D., 473 (Hel.)
—— & Pascale, H., 473 *quin.* (476) (Hel.)
—— & Pestana, B. R., 609, 610, 673, 674 (Leish.)
——, with Villela & Pestana, 610 (Leish.)

Pestana, B. R., with Pessôa, 609, 610, 673, 674 (Leish.)
——, with Villela & Pessôa, 610 (Leish.)
Pettit, V. D., with Diggs, 716 (Haem.)
Pick, F., 314 (Am.)
Pieraerts, G., 99 (Hel.)
Pifano, F., 245, 247 (Tryp.)
Piney, A., 727 (B.R.)
Pinkerton, H., with Henderson & Moore, 645 (Der.)
Pirie, J. H. H. & Grasset, E., 311 (Pl.)
Platts, S. A., with McKenzie, 326 (Diet.)
Plotz, H. & Reagan, R., 589 (Rab.)
—— & Wertman, K., 680 (Typh.)
Plum, D., 687 (Pl.)
Pollitzer, R., Yao, H. Y., Lai, D. G. & Chen, S. K., 159 (Chl.)
Pons, A. R., with Martínez Niochet, 607 (Leish.)
Portuondo Barcelo, J. M., (79) (Y.F.)
Posey, F. M., Jr., with Gilmore & Kean, 796 (Misc.)
Pot, A. W., van Raalte, H. G. S. & van der Sar, A., 552 (Dys.)
Potenza, L., 594 (Mal.)
Prampolini, N., 438 (Mal.)
Prasad, K., 349 (Misc.)
Price, C. H. G., 530 (Bl.)
Priest, R., 738 (Mal.)
Pronk, K. J., 41 (Y. & S.)
Provost, A., with Roubaud, 234 (Tryp.)
Pruis, G. W. A., 52 (Misc.)
Pruys, W. M., 316 (Am.)
Przybylkiewicz, Z., with Eyer & Dillenberg, 385 (Typh.)
Public Health Reports, 115 (Mal.), 303, 762, 763 (Pl.), (350) (Misc.), 611 (Typh.)
Publication of the American Association for the Advancement of Science No. 15, 493 (Misc.)
Purcell, F. M., 785 (Diet.)

Q.

Quattrin, N., 392 (Mal.)
Quevedo, A., 403 (Oph.)

R.

van Raalte, H. G. S., with Pot & van der Sar, 552 (Dys.)
Racine, W., 767 (Lep.)
Raevskii, 253 (Mal.)
——, with Kalabukhov, 307 (Pl.)
Raffaele, G., 263 (Mal.)
Rai, S., 217 (Lep.)
Raja, K. C. K. E., with Lal & Satya Swaroop, 157 (Chl.)
——, with ——, —— & Basak, 157 (Chl.)
——, with Satya Swaroop, Lal & Basak, 157 (Chl.)
Rall, Y. M., 307 (Pl.)
——, Flegontova, A. A. & Sheikina, M. V., 307 (Pl.)
Ram, L. M., 736 (Mal.)
——, with Kumm, 296 (Mal.)
Ramachandra Rao, with Russell, 599 (Mal.)
Ramakrishnan, C. S., with Venkatraman, 458 (Chl.)
——, with —— & Krishnaswami, 160 (Chl.)
Ramamurti, B., 186 (Hel.)
——, with Menon, 477 (Hel.)
Raman, T. K. & Abbu, C., 403 (Oph.)
Ramanamurti, M. V., 186 (Hel.)
Ramirez, E. & Rivero, M. D., 112 (Der.)
Ramon, G., Boquet, P., Richou, R. & Nicol, L., 886 (Vms.)
Ramon, T. K., 251 (Mal.)
Ramos, A. da S., with Corrêa, 737 (Mal.)
—— & Unti, O., 297, (516) (Mal.)
Rand, C. G., 549 (Y.F.)
Rao, B. T. & Menon, T. B., 199 (Misc.)
Rao, M. V. R., 878 (Diet.)
——, with Bopaiya, 895 (Misc.)
Rao, P. K., 415 (Misc.)
Rao, S. S. & Sukhatme, P. V., 185 (Hel.)
Rao, T. R., with Russell, 427 (Mal.)
Rao, V. V., 299 (Mal.)
Rao, W. V. S. K., 186 (Hel.)
Rathje, W., with Oelkers, 767 (Hel.)
Ravenel, B. O., with Beach, 649 (Misc.)
Ray, J. C., Mukerjee, S. & Roy, A. N., 741 (Mal.)
Ray, M., with Hegner, West & Dobler, 124 (Mal.)
Ray, P. N., 186, 188 (Hel.)
Raymond, L. D., 327 (Diet.)
Raymond, W. D., 325 (Diet.)
Read, M., 723 (Misc.)
Read, W. D. B. & Pandit, S. R., 159 (Chl.)
——, —— & Das, P. C., 838 (Chl.)
Reagan, R., with Plotz, 589 (Rab.)
Reardon, L. V., with Chinn, Jacobs & Rees, 765 (Am.)
——, with Cram & Jones, 191 (Hel.)
——, with Rees & Jacobs, (492) (Misc.)
——, with ——, —— & Jones, 28 (Am.)
Reay, E. R., 470 (Hel.)
Rebêlo, A., with de Meillon, 350 (Misc.)
Reed, A. C., 797 (Misc.)
de Reede, C. A., with Mochtar & Rijkebüsch, 347 (Lept.)
Rees, C. W., with Chinn, Jacobs & Reardon, 765 (Am.)
——, Reardon, L. V., Jacobs, L. & Jones, F., 28 (Am.)
——, with —— & ——, (492) (Misc.)
Reichenow, E., 233 (Tryp.)
Reichs-Gesundheitsblatt, 826 (Typh.)
Reid, J. A., with Tweedie & Hodgkin, 522 (Mal.)
Reid, J. D., with Shaw, 104 (Der.)
Reksodiwirjo ,437 (Mal.)
Renaudet, R., with Deschiens (93) (Hel.)
Renault, L. & Versiani, W., 5 (Hel.)
Reviriego, A. J., 218 (Lep.)
Revista de Combate à Lepra, 206 (B.R.)
Rey-Matiz, H., 174 (Leish.), (247) (Tryp.)
Reynes, V. & Richard, J., 377 (Typh.)
Ribeiro, E. B., with Campos, 222 (Lep.)
Riccardi, B., with Torrealba, 248 (Tryp.)
Rice, M. E., with Watson, 436 (Mal.)
Richard, J., with Reynes, 377 (Typh.)
Richardson, D. T., 65 (Y.F.)
Richou, R., with Ramon, Boquet & Nicol, 886 (Vms.)

Riddle, J. W., Spies, T. D. & Hudson, N. P., 339 (Pel.)
Rijkebüsch, with Mochtar & de Reede, 347 (Lept.)
Rimington, C., 529 (Bl.)
Rin, S., 298 (Mal.)
Riou, M., 50, 413 (Misc.)
Risquez, J. R. & Boza, F. V., 560 (Hel.)
Ristorcelli, A. & Dao Van Ty, (833) (Fev.)
Rivas, C. I. & Góbich, E., (706) (Hel.)
Rivera, E. M., with Doull, Saunders, Guinto & Morales, 219 (Lep.)
Rivero, M. D., with Ramirez, 112 (Der.)
Ro, M. & Yokogawa, S., 13 (Hel.)
Robertson, R. C., 11 (Hel.), 649 (Misc.)
——, Bard, S. & Chen, J., (334) (Bb.)
—— & Chang, T. L., (177) (Mal.)
Robinson, G. G., 692, 847 (R.F.)
——, with Cole & Cosgrove, 746 (Leish)
Robinson, P., 279 (Misc.), 846 (R.F.)
Robson, J. M. & Scott, G. I., 402 (Oph.)
Rocha, A., with Magalhães, 452 (Typh.)
Rockefeller Foundation, 64, 615 (Y.F.), 140 (Typh).
Rodriguez, J. N., with Guinto, 216, 217 (Lep.)
Rodriguez Molina, R., 638 (Sp.)
Rodriguez Olleros, A., 715 (Sp.)
Roe, M. A., Wilcox, A. & Lillie, R. D., 484 (Misc.)
Rogers, L., (22) (Mal.)
—— & Megaw, J. W. D., 282 (B.R.)
Rogliano, A. G., 694 (R.F.)
Roig y Raventós, J., 608 (Leish.)
Romaña, C., 292 (Mal.)
Romero Escacena, G., 139 (Typh.)
——, Bolinches, J. & Alvarez, M., 540 (Typh.)
van Rooyen, C. A., 17 (Mal.)
Röpke, F., 722 (Misc.)
Ros, A., with Carr, Meléndez, A. F. & Meléndez, J. F., 515 (Mal.)
Rose, 372 (Fev.), 396 (Mal.)
Rose, H. M., with Culbertson, 95 (Hel.)
Rosenfeld, G. & Cardoso, F. A., 247 (Tryp.)
Rosenfeld, S. & Rubinstein, J., 405 (Vms.)
Rosenthal, L. H., with Weidman, 106 (Der.)
Rosinger, Z., 738 (Mal.)
Ross, G. R. & Aylen, D., 260 (Mal.)
Rossas, T. P., 218 (Lep.)
Rossiysky, F., 142 (Typh.)
Rottenstein, H. S., with Lee & Chao, 402 (Oph.)
Roubaud, E. & Provost, A., 234 (Tryp.)
Rouse, A. H., with Myers, 37 (Dys.)
Roussel, H., with Estobitte, 888 (Oph.)
Roy, A. N., with Ray & Mukerjee, 741 (Mal.)
Roy, B., with Ghosh, 341 (Diet.)
Roy, B. C., 531 (Bl.)
Roy, D. N., with Chopra & Ghosh, 260 (Mal.)
—— & Ghosh, S. M., 900 (Misc.)
——, —— & Chopra, R. N., 795 (Misc.)
—— & Roy, P. C., 437 (Mal.)
——, with Strickland, 578 (Misc.)
Roy, P. C., with Roy, 437 (Mal.)
Roy, R., with Barma & Bhattacharya, (620) (Am.)
Rozeboom, L. E., 74 (Y.F.), 811 (Mal.)
——, Fox, L. A. & Laird, R. L., 430 (Mal.)
—— & Gabaldon, A., (431) (Mal.)
Rozeboom, L. E. & Laird, R. L., 664 (Mal.)
Ruangsiri, C., with Cowdry, 233 (Lep.)
Rubinstein, J., with Rosenfeld, 405 (Vms.)
Ruchelli, A. P., (672) (Tryp.)
Rudolf, E., 228 (Lep.)
Ruffin, J. N., with Schulze, 839 (Am.)
Ruiz, H., with Kumm, 290 (Mal.)
——, with ——, & Volio, 291 (Mal.)
Ruiz Nazario, R., with Carrión & Hernandez Morales, 110 (Der.)
Runacher, A., with Anderson, Berge & Fauconnier, 342 (R.F.)
Runner, A. G., 307 (Pl.)
Russell, B. A. S., 482 (Haem.)
Russell, H., 165 (Chl.)
Russell, P. F., 435, 603 (Mal.)
—— & Knipe, F. W., 667 (Mal.)
—— & Menon, M. K., 808 (Mal.)
—— & Mohan, B. N., 117, 391 (Mal.)
——, Mulligan, H. W. & Mohan, B. N., 300 (Mal.)
——, with Mulligan & Mohan, 300 (Mal.)
—— & Ramachandra Rao, T., 598, 599 (Mal.)
—— & Rao, T. R., 427 (Mal.)
Rustomjee, K. J., with Sivalingham, 663 (Mal.)
Rutter, A. G., (39) (Y. & S.)

S.

Sablin, P. E. & Egorova, O. G., 519 (Mal.)
Sagher, F., with Dostrovsky, 749 (Leish.)
Saldun de Rodriguez, M. L., Soto, J. A. & Giampietro, J., 704 (Hel.)
Salgar, A. A., (706) (Hel.)
Salica, P. N., with Mazza & Freire, 671 (Tryp.)
—— P. R., with Mazza, 248 (Tryp.)
Salis, H., 28 (Am.)
Salt, G., (351) (Misc.)
Sanders, J. P., 813 (Mal.)
Sandes, J. D., 316 (Am.)
Sandground, J. H., 7 (Hel.)
Saner, R. G., with Gear & Harris, 830 (Typh.)
Sanyal, S. N., (159) (Chl.)
Sapero, J. J., 472 (Hel.)
de Saram, G. S. W. & Gunewardene, S. R., 570 (Hel.)
Sarkar, S. K., 297 (Mal.)
Sarkar, S. N., 711 *bis*, 712 (Diet.)
Sati, M. H., 747 (Leish.)
Satya Swaroop, with Lal & Raja, 157 (Chl.)
——, with ——, —— & Basak, 157 (Chl.)
——, Raja, K. C. K. E., Lal, R. B. & Basak, K. C., 157 (Chl.)
Saunders, G., 534 (Tryp.)
Saunders, G. F. T., 532 (Tryp.)
Saunders, G. M., 182 (Hel.)
—— & Bancroft, H., 898 (Misc.)
——, with Doull, Rivera, Guinto & Morales, 219 (Lep.)
Savoor, S. R. & Lewthwaite, R., 345 (R.B.F.)
Sawitz, W., 771 (Hel.)
—— & Faust, E. C., 765 (Am.)
Sawyer, W. A., 683 (Y.F.)
Schaeffer, C. O., 197 (Misc.)
Scharff, J. W. & Tweedie, M. W. F., 814 (Mal.)

Schenone, B. H., with Grana, 705 (Hel.)
Schlegel, B., 351 (Misc.)
Schleicher, E. M., 786 (Haem.)
Schmidt, G., 280 (Misc.)
Schmidt, H., Christian T. T. & Smotherman, W. M., 465 (Hel.)
——, with Kikuth, 450 (Leish.)
Schneider, O., 489 (Misc.)
Schnitzer, R. J., Siebenmann, C. & Bett, H. D., 862 (Hel.)
Schüffner, W., 431 (Mal.), 847 (Lept.)
Schujman, S., 225 (Lep.)
Schulemann, W., 743 (Mal.)
—— & Knoche, E., 25 (Mal.)
Schulze, W. & Ruffin, J. N., 839 (Am.)
Schumacher, W., with Seck, 776 (Hel.)
Schwartz, L., Furst, W. & Flippin, H. F., 601 (Mal.)
Schwarz, J. & Straub, M., (709) (Hel.)
Scolari, P. G. & Ocaña, T., 565 (Hel.)
Scott, G. I., with Robson, 402 (Oph.)
Scott, H. H., 798 (B.R.)
Scott, J. A., 865 (Hel.)
Scott, R. R., (653) (Misc.)
Seaton, D. R. & Lumsden, W. H. R., 490 (Misc.)
Seck, P. & Schumacher, W,, 776 (Hel.)
Sédan, J., Koutseff, A., Livon, J. & Gauran, M., 888 (Oph.)
Seetharama Iyer, P. V., Shortt, H. E. & Menon, K. P., 524 (Mal.)
——, with —— & ——, 670 (Mal.)
Segal, L. J., 568 (Hel.)
Séguy, E., (796) (Misc.)
Seiffert, G., 611 (Typh.)
Sellards, A. W., 78 (Y.F.)
Sellers, T. F., with Damon, 590 (Rab.)
Sen, B., with Chopra & Gupta, 255 (Mal.)
Sen, G., with Napier & Kirwan, 171 (Leish.)
Sen, N. R., with Lowe & Dharmendra, 557 (Lep.)
Sen, P., 294, 664, 810 (Mal.)
Senekji, H. A., 93 (Hel.), 174 (Leish.)
—— & Zebouni, N., 167 (Leish.)
Sen Gupta, P. C., with Greval & Das, 225 (Lep.)
Senra, J. de M., 698 (Hel.)
Sequiera, J. H. & Dowdeswell, R. M., 796 (Misc.)
Seurat, L. G., (604) (Mal.)
Shah, M. H., 173 (Leish.)
Shaw, F. W. & Reid, J. D., 104 (Der.)
Shaw, G. W. B. & Clyne, A. J., 14 (Hel.)
Sheikina, M. V., with Fenyuk, 308 (Pl.)
——, with Rall' & Flegontova, 307 (Pl.)
Sheldon, A. J., with Brown, 190 (Hel.)
Shelton, C. F., 76 (Y.F.)
Shillong, 83 (Rab.), 161 (Chl.)
Shimizu, S., with Ishii & Tsuda, 126 (R.F.)
Shortt, H. E., Menon, K. P. & Seetharama Iyer, P. V., 670 (Mal.)
——, with Seetherama Iyer & Menon, 524 (Mal.)
Shterngol'd, E., with Khodukin, 116 (Mal.)
Shute, P. G., with Nicol, 739 (Mal.)
Sicari, S., with d'Alessandro, 256 (Mal.)
Sicart, M., (516) (Mal.)
Siebenmann, C., with Schnitzer & Bett, 862 (Hel.)
Siegenbeek van Heukelom, A. & Wahab, 257 (Mal.)
Siliceo Ambía, L., 620 (Am.)
Silva, F., 114 (Der.)
de Silva, K. J. L., 890 (Oph.)
Silva, R., with Castaneda, 151 (Typh.)
de Silva, S., 414 (Misc.)
Silvetti, L. M., with Lobe, 305 (Pl.)
Silvey, W. J., with Jordan, 122 (Mal.)
Singh, B., 833 (Fev.)
Sinjovich, U. & Somaini, A. F., 246 (Tryp.)
Sivalingham, V. & Rustomjee, K. J., 663 (Mal.)
Sliwensky, M., 633 (Hel.)
Sloan, T. B. M. & Ebenezer, R., 559 (Lep.)
de Smidt, F. P. G., 688 (Pl.)
Smiles, J., with Hawking, 238 (Tryp.)
Smith, E. C., 686 (Y.F.)
Smith, G. E., 811 (Mal.)
——, Watson, R. B. & Crowell, R. L., 392 (Mal.)
Smith, H. H., 65 (Y.F.)
——, Calderón-Cuervo, H. & Leyva, J. P., 77 (Y.F.)
Smith, J., 128 (Lept.)
Smith, J. E., with Julianelle, 887 (Oph.)
Smith, L. M., 105 (Der.)
Smith, R. O. A. & Ahmed, I., 448 (Leish.)
———, Halder, K. C. & Ahmed, I., 447, 449 (Leish.)
Smithburn, K. C., with Mahaffy, Hughes & Kirk, 456 (Y.F.)
—— with Mahaffy, Jacobs & Gillett, 759 (Y.F.)
Smotherman, W. M., with Schmidt & Christian, 465 (Hel.)
Snapper, I., 726 (B.R.)
Sneath, P. A. T., 203 (Misc.)
Snyder, J. C. & Anderson, C. R., 374 (Typh.)
Snyman, P. S., 86 (Rab.)
So, K., 298, 299 (Mal.)
So, T., with Akashi, 394 (Mal.)
Soares, H. de A., 293 (Mal.)
Soedarsono, 599 (Mal.)
Soesman, J., 325 (Dys.)
Soetopo, M., 39 (Y. & S.)
——, with Tumbelaka, 39 (Y. & S.)
Sokhey, S. S., 300 (Pl.)
Solomon, H. C., with Kopp, 665 (Mal.)
Somaini, A. F., with Sinjovich, 246 (Tryp.)
van Someren, E. C. C., 73 (Y.F.)
Sompayrac, L. M., with Millspaugh, 774 (Hel.)
Soni, R. L., 614 (Typh.)
Soong, H. Y. & Anderson, H. H., 171 (Leish.)
Soper, F. L., 548 (Y.F.)
Sorge, G., 22 (Mal.)
Soriano, L. J., with Africa, & Dy, 27 (Mal.)
Sorol, .R V., 415 (Misc.)
Sory, R., 887 (Oph.)
Sosa Gallardo, J., with de Villafañe Lastra, & Fernando Videla, 309 (Pl.)
Soto, J. A., with Saldun de Rodriguez & Giampietro, 704 (Hel.)
Sotolongo, F. & Goldberger, J., 647 (Misc.)
Souter, A. W., with Kark & Hayward, 45 (Sp.)
Southwell-Sander, G. & Hand, R. F., 393 (Mal.)

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